MEDICO-CHIRURGICAL TRANSACTIONS.

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THE RO[illegible]

MEDICAL AND CH[illegible]

LONDON

VOLUME THE FIFT[illegible]

LONDON:
LONGMANS, GREEN, READER [illegible]
PATERNOSTER ROW.

1878.

MEDICO-CHIRURGICAL TRANSACTIONS.

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THE ROYAL

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OF

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1873.

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ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

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FOR THE SESSION OF 1873-74.

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RINGER, SYDNEY, M.D.
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SIMON, JOHN, D.C.L., F.R.S.
SMITH, SPENCER.
SMITH, THOMAS.
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THOMPSON, REGINALD, M.D.
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WEST, CHARLES, M.D.
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1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
1815. HENRY CLINE.
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, Bart., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY.
1825. GEORGE BIRKBECK, M.D.
1827. BENJAMIN TRAVERS.
1829. PETER MARK ROGET, M.D.
1831. SIR WILLIAM LAWRENCE, Bart.
1833. JOHN ELLIOTSON, M.D.
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1839. SIR BENJAMIN COLLINS BRODIE, Bart., D.C.L.
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1849. THOMAS ADDISON, M.D.
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1861. BENJAMIN GUY BABINGTON, M.D.
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1865. SIR JAMES ALDERSON, M.D.
1867. SAMUEL SOLLY.
1869. GEORGE BURROWS, M.D.
1871. THOMAS BLIZARD CURLING.
1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.

FELLOWS

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON.

EXPLANATION OF THE ABBREVIATIONS.

P.—President.	V.P.—Vice-President.
T.—Treasurer.	S.—Secretary.
L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1873.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital; 13, Suffolk-square, Cheltenham.

1851 *ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

1842 †ACTON, WILLIAM, 17, Queen Anne Street, Cavendish square. *Trans.* 1.

Elected

1852 ADAMS, WILLIAM, Consulting Surgeon to the National Orthopædic Hospital, Great Portland Street; 5, Henrietta street, Cavendish square. C. 1873. *Trans.* 2.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 38, Park square, Leeds. *Trans.* 3.

1869 ALLEN, PETER, M.D., Aural Surgeon to St. Mary's Hospital; 117, Harley street, Cavendish square.

1863 ALTHAUS, JULIUS, M.D., Physician to the Infirmary for Epilepsy and Paralysis; 18, Bryanston street, Portman square. *Trans.* 2.

1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1867 ANSTIE, FRANCIS EDMUND, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 16, Wimpole street, Cavendish square.

1870 ARNOTT, HENRY, Assistant-Surgeon to St. Thomas's Hospital; 6, Nottingham place, Marylebone road.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

1817 †ASHBURNER, JOHN, M.D., F.L.S., 161A, Piccadilly. C. 1821, 1830-31.

1851 ASHTON, THOMAS JOHN, Consulting Surgeon to the St. Marylebone Infirmary; 31, Cavendish square.

Elected

1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital; 20A, Temple row, Birmingham.

1865 BAKER, WILLIAM MORRANT, Assistant Surgeon to, and Lecturer on Anatomy and Physiology, and Warden of the College at, St. Bartholomew's Hospital. *Trans.* 2.

1869 BAKEWELL, ROBERT HALL, M.D., New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Deputy Inspector-General of Hospitals Principal Medical Officer, Royal Victoria Hospital, Netley, Southampton. C. 1852-3. V.P. 1860-1. T. 1872. *Trans.* 2.

1848 BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 7, Compton terrace, Islington. C. 1872. *Trans.* 5.

1849 BALLARD, THOMAS, M.D., 10, Southwick place, Hyde park square.

1866 *BANKS, JOHN THOMAS, M.D., Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; 10, Merrion square east, Dublin.

1847 BARCLAY, ANDREW WHYTE, M.D., *Vice-President*, Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Trans.* 2.

1862 BARKER, EDGAR, jun., 21, Hyde park street.

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. Thomas's Hospital; Examiner in Midwifery at the University of London; 31, Grosvenor street. *Trans.* 4.

Elected

1864 Barratt, Joseph Gillman, M.D., 8, Cleveland gardens, Bayswater.

1840 Barrow, Benjamin, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 Barwell, Richard, Surgeon to, and Lecturer on Anatomy and Clinical Surgery at, the Charing Cross Hospital; 32, George street, Hanover square. *Trans.* 1.

1844 Basham, William Richard, M.D., Senior Physician to the Westminster Hospital; 17, Chester street, Belgrave square. S. 1852-4. C. 1860-1. V.P. 1864-5. T. 1871. *Trans.* 2.

1868 Bastian, Henry Charlton, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1862 Beale, Lionel Smith, M.B., F.R.S., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. *Trans.* 1.

1860 *Bealey, Adam, M.D., M.A., Camb., Oak Lea, Harrogate.

1841 †Beaman, George, M.D., 3, Henrietta street, Covent garden.

1856 Beardsley, Amos, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1836 Beaumont, William Rawlings, Consulting Surgeon to the Toronto General Hospital; Toronto, Canada West. *Trans.* 3.

1871 Beck, Marcus, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.

1858 Begley, William Chapman, A.M., M.D.

1819 †Bell, Thomas, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

Elected

1871 Bellamy, Edward, Senior Assistant-Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon to the Royal Infirmary for Children and Women, Waterloo road; 59, Margaret street, Cavendish square.

1847 Bennet, James Henry, M.D., The Ferns, Weybridge, and Mentone.

1845 Berry, Edward Unwin, 76, Gower street, Bedford square.

1820 Bertin, Stephen, Paris.

1872 Beverley, Michael, M.D., 63, St. Giles's street, Norwich.

1865 *Bickersteth, Edward Robert, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1815 †Billing, Archibald, M.D., F.R.S., Member of the Senate of the University of London; 34, Park lane. C. 1825. V.P. 1828-9.

1854 Bird, Peter Hinckes, F.L.S., 4, Clifton Terrace, Lytham, Lancashire.

1856 Bird, William, Bute House, Hammersmith.

1849 Birkett, Edmund Lloyd, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6.

1851 Birkett, John, F.L.S., *Treasurer*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Examiner in Surgery at the University of London; 59, Green Street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-73. *Trans.* 8. *Sci. Com.*

1866 Bishop, Edward, M.D., Cintra park, Upper Norwood.

1843 Black, Patrick, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square, C. 1856. V.P. 1866. T. 1869-70.

1840 Blakiston, Peyton, M.D., F.R.S., Victoria street, Pimlico.

Elected

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street.

1867 BLOXAM, JOHN ASTLEY, Assistant-Surgeon to Charing Cross Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 BOJANUS, LOUIS HENRY, M.D., Wilna.

1846 BOSTOCK, JOHN ASHTON, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 54 Chester square, Belgravia. C. 1861-2. V.P. 1870-71. *Sci. Com.*

1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City.]

1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.

1841 †BOWMAN, WILLIAM, F.R.S., F.L.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.

1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Joint Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Trans.* 1.

1851 BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Orthopædic Department of, and Lecturer on Orthopædic Surgery at, St. George's Hospital, and Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Trans.* 2. *Pro.* 1.

Elected

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 56, Curzon street, Mayfair.

1844 †BROOKE, CHARLES, M.A., F.R.S., Consulting Surgeon to the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-72.

1857 *BROWN, ROBERT, Surgeon to the Cumberland Infirmary, 5, Devonshire street, Carlisle.

1860 BROWN-SEQUARD, CHARLES EDOUARD, M.D., F.R.S., late Professor of Physiology and Pathology, Massachusetts Medical College, Harvard University, Boston, U.S.; Rue Gay-Lussac, 28, Paris. *Sci. Com.*

1867 BRUNJES, MARTIN, 42, Brook street, Grosvenor square.

1871 BRUNTON, THOMAS LAUDER, M.D., Casualty Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; 23, Somerset street, Portman square.

1860 BRYANT, THOMAS, Surgeon to Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873. *Trans.* 8; *Pro.* 1. *Sci. Com.*

1855 BRYANT, WALTER JOHN, L.R.C.P. Edinb.; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., Inspector, Medical Department, Local Government Board; 24, Nottingham place, Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D. [Sir Francis Smith's, 15, Thurloe place, South Kensington].

1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich; Ashleigh, Barnstaple. C. 1846-7. V.P. 1857. *Trans.* 5.

1833 †BURROWS, GEORGE, M.D., D.C.L., F.R.S., President of the Royal College of Physicians; Physician Extraordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V.P. 1849-50. P. 1869-70. *Trans.* 2.

Elected

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.

1873 BUTLIN, HENRY TRENTHAM, Surgical Registrar to St. Bartholomew's Hospital; Registrar of the Hospital for Sick Children; 13, Jewin crescent, Aldersgate street.

1871 BUTT, WILLIAM F., 12, South street, Park lane.

1818 BUTTER, JOHN, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villa, Plymouth.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1861 CALLENDER, GEORGE WILLIAM, F.R.S., Surgeon to, and Joint-Lecturer on Surgery at, St. Bartholomew's Hospital; Lecturer on Anatomy and Physiology at the Royal College of Surgeons; 47, Queen Anne street, Cavendish square. *Trans.* 4. *Sci. Com.*

1852 *CANNEY, GEORGE, M.D., Bishop-Auckland, Darlington, Durham.

1847 CARLILL, JOHN BURFORD, M.D., 42, Weymouth street, Portland place.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 69, Wimpole street, Cavendish square, W.

1845 CARTWRIGHT, SAMUEL, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*

Elected

1868 CAVAFY, JOHN, M.D., Demonstrator of Histology and Lecturer on Comparative Anatomy at St. George's Hospital Medical School; Assistant-Physician to the Victoria Hospital for Children; 13, Arlington street, Piccadilly.

1871 CAYLEY, WILLIAM, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the London Fever Hospital; 58, Welbeck street, Cavendish square.

1845 CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 24, Mount street, Grosvenor square. C. 1861. V.P. 1867. L. 1869-72. *Trans.* 1.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1837 †CHAPMAN, HENRY THOMAS, 21, Lower Seymour street, Portman square. C. 1858.

1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. Mary's Hospital; Assistant-Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLAM SELBY, M.D., Assistant-Physician to, and Lecturer on Comparative Anatomy at, St. Bartholomew's Hospital; 2, Upper George street, Bryanston square.

Elected

1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P. 1855-6. *Trans.* 4.

1848 CLARKE, JOHN, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Physician to the General Lying-in Hospital; 42, Hertford street, Mayfair. C. 1866.

1866 CLARKE, WILLIAM FAIRLIE, M.A. (Oxon.), Assistant-Surgeon to the Charing Cross Hospital; 12, Mansfield street, Cavendish square. *Trans.* 1.

1861 *CLARKE, WILLIAM JAMES, Surgeon to the Huddersfield Infirmary; John-William street, Huddersfield, Yorkshire.

1850 CLARKSON, JOSIAH, New Hall street, Birmingham. *Trans.* 1.

1842 †CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square. C. 1873.

1857 COATES, CHARLES, F.R.C.P. Edinb., Physician to the Bath United General Hospital; 10, Circus, Bath.

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 7, Suffolk place, Pall mall. *Trans.* 2.

1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.

1818 COLE, ROBERT, F.L.S., Holybourne, Hampshire.

1854 COLLINS, FREDERICK, M.D., Wanstead Lodge, Essex.

Elected

1865 COOPER, ALFRED, Surgeon to the Royal Hospital for Diseases of the Chest, Additional Surgeon for Out-patients to the Lock Hospital; Assistant-Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square, W.

1819 COOPER, GEORGE, Brentford, Middlesex.

1841 †COOPER, GEORGE LEWIS, one of the Surgeons to the National Vaccine Institution, and Teacher of Vaccination to the Medical School of University College; Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square. C. 1860-1. *Trans.* 1.

1843 COOPER, WILLIAM WHITE, *Vice-President*, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.-P. 1873.

1835 COPELAND, GEORGE FORD, 5, Bayshill villas, Cheltenham.

1868 CORNISH, WILLIAM ROBERT, Surgeon, Madras Army; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 9, Clarendon place, Belfast.

1839 *CORSELLIS, CHARLES CÆSAR, M.D., F.L.S., Benson, Oxon.

1853 CORY, WILLIAM GILLETT, M.D.

1847 †COTTON, RICHARD PAYNE, M.D., Physician to the Hospital for Consumption, Brompton; 33, Cavendish square. C. 1863.

1828 †COULSON, WILLIAM, F.L.S., Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 2, Frederick's place, Old Jewry, and 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 29, St. James's place.

Elected

1860 †COUPER, JOHN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street.

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Assistant-Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 19, George street, Hanover square.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Wiesbaden. C. 1853-4.

1868 CRAWFORD, THOMAS, M.D., Deputy Inspector-General of Hospitals (India); Umbalah, Punjaub.

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1847 CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Trans.* 1.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 61, Brook street, Grosvenor square.

1862 CROMPTON, SAMUEL, M.D., Physician to the Salford Royal Hospital and Dispensary; 24, St. Ann's square, Manchester.

1837 CROOKES, JOHN FARRAR, 5, Waterloo crescent, Dover.

1860 CROSS, RICHARD, M.D., 5, Queen street, Scarborough.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1851 CUMMING, JAMES CAMERON, M.D.

1865 CURGENVEN, J. BRENDON, 11, Craven hill gardens, Bayswater.

1846 CURLING, HENRY, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seaman's Infirmary; Ramsgate, Kent.

Elected

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Trans.* 13. *Pro.* 1. *Sci. Com.*

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

1852 CUTLER, THOMAS, M.D., Spa, Belgium.

1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 79, Grosvenor street. *Trans.* 1.

1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1846 DAVIES, FREDERICK, M.D., 124, Gower street, Bedford square. C. 1873.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 18, Gay street, Bath.

1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 24, Harley street, Cavendish square. C. 1869-70.

1818 DAWSON, JAMES, Wray Castle, Windermere.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1867 DE MERIC, VICTOR, Surgeon to the Royal Free Hospital, and to the German Hospital, Dalston; 52, Brook street, Grosvenor square.

1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859 DICKINSON, WILLIAM HOWSHIP, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. George's Hospital; Physician to the Hospital for Sick Children; Examiner at the Royal College of Physicians, and at the University of Cambridge; 11, Chesterfield street, Mayfair. *Trans.* 12. *Sci. Com.*

Elected

1844 DICKSON, ROBERT, M.D., F.L.S. C. 1860.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting-Ophthalmic Surgeon to the Asylum for Idiots; 29, Lower Seymour street, Portman square. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Trans.* 4.

1862 DOBELL, HORACE B., M.D., Physician to the Royal Hospital for Diseases of the Chest, City road; 84, Harley street. *Trans.* 1.

1845 DODD, JOHN.

1857 DOUGLAS, ARCHIBALD, M.D., 8, Clifton place, Sussex square, Hyde park.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 39, Welbeck street, Cavendish square. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1853 DRUITT, ROBERT, M.R.C.P. India. [Mr. Thorn's, 3, Little Stanhope street, Mayfair.] *Trans.* 2.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 99, Southampton row, Russell square.

1865 DUCKWORTH, DYCE, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1845 DUFFIN, EDWARD WILLSON, 18, Devonshire street, Portland place. *Trans.* 1.

1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham Common.

1871 *DUKES, CLEMENT, M.B. and B.S., Horton crescent, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Canterbury road, Thornton Heath.

1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

Elected

1861 Du Pasquier, Claudius Francis, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales; 62, Pall Mall.

1863 Durham, Arthur Edward, F.L.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 82, Brook street, Grosvenor square. *Trans.* 5. *Sci. Com.*

1843 Durrant, Christopher Mercer, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 Dyer, Henry Sumner, M.D., Sennowe Hall, Guist, Norfolk. C. 1854-5.

1872 Eager, Reginald, M.D., Assistant Medical Superintendent of the Buckinghamshire Lunatic Asylum, Stone.

1836 Earle, James William, late of Norwich.

1868 Eastes, George, M.B., Lond., Surgeon-Accoucheur to the Western General Dispensary; 5, Albion place, Hyde park square.

1824 Edwards, George.

1823 Egerton, Charles Chandler, Kendall Lodge, Epping.

1869 Elam, Charles, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.

1861 *Elliot, Robert, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle; 35, Lowther street, Carlisle.

1848 Ellis, George Viner, Professor of Anatomy in University College, London. C. 1863-4. *Trans.* 2.

1868 Ellis, James, M.D., Belle Grove villa, Welling, Blackheath, and Infirmary, St. Pancras Workhouse.

1854 *Ellison, James, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †Erichsen, John Eric, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. *Trans.* 2.

1845 Evans, William Julian, M.D., Pinner, Middlesex.

Elected

1864 FAGGE, CHARLES HILTON, M.D., Assistant-Physician to, and Lecturer on Pathology at, Guy's Hospital; and Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Trans.* 4.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., Lynton, North Devon.

1858 FALCONER, RANDLE WILBRAHAM, M.D., Physician to the Bath United Hospital; 22 Bennett street, Bath.

1862 FARQUHARSON, ROBERT, M.D., 23, Brook street, Grosvenor square.

1844 FARRE, ARTHUR, M.D., F.R.S., Physician-Accoucheur to H.R.H. the Princess of Wales; 12, Hertford street, Mayfair. C. 1857. V.P. 1864. *Sci. Com.*

1872 FAYRER, JOSEPH, M.D., F.R.S. Ed., Honorary Physician to the Queen; Surgeon-Major, Bengal Army; Professor of Surgery, Medical College, Calcutta. [10A, Stanhope place, Hyde park.]

1872 FENWICK, JOHN C. J., M.B., 30, Devonshire street, Portland place.

1863 FENWICK, SAMUEL, M.D., Assistant-Physician to, and Lecturer on Histology at, the London Hospital; 29, Harley street, Cavendish square. *Trans.* 3.

1841 †FERGUSSON, SIR WILLIAM, Bart., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon to King's College Hospital; 16, George street, Hanover square. C. 1849-50. V.P. 1863-4. *Trans.* 4.

1852 *FIELD, ALFRED GEORGE, Alverton Manor House, Stratford-on-Avon.

1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1866 FISH, JOHN CROCKETT, B.A., M.B., Camb., Junior Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1836 †FISHER, SIR JOHN WILLIAM, 33, Park Lane. C. 1843-4.

Elected

1860 FITZGERALD, THOMAS GEORGE, Surgeon-Major; 6, Savile row, Burlington gardens.

1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to the Western General Dispensary, 30, Sussex gardens, Hyde park.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Physician to the Birmingham General Hospital.; 7, Waterloo street, Birmingham. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1848 FORBES, JOHN GREGORY, 82, Oxford Terrace, Hyde Park. C. 1868-9. *Trans.* 3.

1852 †FORSTER, JOHN COOPER, *Secretary,* Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 29, Upper Grosvenor street. C. 1868-9. S. 1873. *Pro.* 1.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.

1859 FOX, EDWARD LONG, M.B., Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church house, Clifton, Gloucestershire.

1858 FOX, WILSON, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. *Trans.* 3.

1871 FRANK, PHILIP, M.D., Cannes, France.

1843 FRASER, PATRICK, M.D. C. 1866.

1868 FREEMAN, WILLIAM HENRY, 29, Spring gardens.

1836 †FRENCH, JOHN GEORGE, late Surgeon to the St. James's Infirmary; 10, Cunningham place, Maida hill. C. 1852-3.

1849 FRERE, ROBERT TEMPLE, M.A., F.R.C.P., 143, Harley street.

Elected

1846 Fuller, Henry William, M.D,, Physician to St. George's Hospital; 13, Manchester square. C. 1862. S. 1864-5. V.P. 1868-9. *Trans.* 3.

1864 *Gairdner, William Tennant, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.

1865 Gant, Frederick James, Surgeon to the Royal Free Hospital, 16, Connaught square, Hyde park. *Trans.* 2.

1867 Garland, Edward Charles, L.R.C.P. Edin., Yeovil, Somerset.

1867 Garlike, Thomas, W., Tulse Hill, Brixton.

1854 Garrod, Alfred Baring, M.D., F.R.S., Professor of Materia Medica in King's College, London, and Physician to King's College Hospital; Examiner in Materia Medica at the University of London; 10, Harley street, Cavendish square. C. 1867. *Trans.* 8.

1857 Gascoyen, George Green, Surgeon to the Lock Hospital; Assistant-Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 48, Queen Anne street, Cavendish square. S. 1866-9. C. 1871-2. *Trans.* 3. *Sci. Com.* 2.

1851 Gaskoin, George, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne Park.

1819 Gaulter, Henry.

1848 Gay, John, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south.

1866 Gee, Samuel Jones, M.D., Assistant-Physician to St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square.

1821 *George, Richard Francis, 20, Marlborough buildings, Bath.

1870 Godson, Clement, M.B., C.M., Physician to the Samaritan Free Hospital; 8, Upper Brook street, Grosvenor square.

Elected

1867 Goodeve, Edward, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.

1851 Goodfellow, Stephen Jennings, M.D., Consulting Physician to the Middlesex Hospital; 5, Savile row, Burlington gardens. C. 1864-5. *Trans.* 2.

1851 Gowlland, Peter Yeames, Surgeon to St. Mark's Hospital; 34, Finsbury Square.

1844 Grantham, John, Crayford, Kent.

1846 Gream, George Thompson, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; 2, Upper Brook street, Grosvenor square. C. 1863.

1868 Green, T. Henry, M.D., Senior Assistant-Physician to, and Lecturer on Pathology at, Charing Cross Hospital; 74, Wimpole street, Cavendish Square.

1843 Greenhalgh, Robert, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 72, Grosvenor street. C. 1871-2.

1860 Greenhow, Edward Headlam, M.D., F.R.S., Physician to, and Lecturer on the Practice of Medicine at, the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 14a, Manchester square. *Trans.* 3.

1868 Grigg, William Chapman, M.D., Medical Officer, Queen Charlotte's Lying-in-Hospital; 6, Curzon street, Mayfair.

1814 Grove, John, M.D., Salisbury.

1852 Grove, John, Spring Grove, Hampton, Middlesex.

1860 Gueneau de Mussy, Henri, M.D., late Physician to the French Hospital, Lisle street, Leicester square; 15, Rue du Cirque, Paris.

1849 Gull, Sir William Withey, Bart., M.D., D.C.L., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. *Trans.* 4.

Elected

1837 GULLY, JAMES MANBY, M.D., Great Malvern, Worcestershire.

1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. *Trans.* 3.

1849 HAILEY, HAMMETT, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.

1848 HALLEY, ALEXANDER, M.D., F.G.S., 16, Harley street, Cavendish square.

1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.

1838 †HANCOCK, HENRY, Consulting Surgeon to the Charing Cross Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 76, Harley street, Cavendish square. C. 1851. V.P. 1869.

1836 HARDING, JOHN FOSSE, Mount Sandford, Southborough, Tunbridge Wells. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873.

1857 HARLEY, GEORGE, M.D., F.R.S., 25, Harley street, Cavendish square. C. 1871-2. *Trans.* 1. *Sci. Com.* 2.

1864 HARLEY, JOHN, M.D., F.L.S., Assistant-Physician to, and Joint Lecturer on Physiology at, St. Thomas's Hospital; 78, Upper Berkeley street, Portman square. *Trans.* 5.

1866 HARPER, PHILLIP H., 30, Cambridge street, Hyde park.

1859 HARRIS, FRANCIS, M.D., F.L.S., Physician to St. Bartholomew's Hospital; 24, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Professor of Midwifery and Diseases of Women and Children, Madras Medical College; 31, Devonshire street, Portland place.

1870 HARRISON, REGINALD, Assistant Surgeon to the Liverpool Royal Infirmary, and Lecturer on Anatomy at the School of Medicine; 51, Rodney street, Liverpool.

Elected

1841 †HARVEY, WILLIAM, Surgeon to the Royal Dispensary for Diseases of the Ear and to the Freemasons' Female Charity; Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

1854 HAVILAND, ALFRED, Medical Officer of Health.

1870 HAWARD, J. WARRINGTON, Assistant-Surgeon to the Hospital for Sick Children; 46, Queen Anne Street, Cavendish square. *Trans.* 1.

1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.

1848 HAWKSLEY, THOMAS, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 6, Brook street, Grosvenor square.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square.

1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, Worcestershire.

1848 *HEALE, JAMES NEWTON, M.D., Winchester, Hants.

1865 HEATH, CHRISTOPHER, Surgeon to University College Hospital, and Lecturer on Operative Surgery in University College, London; 9, Cavendish place, Cavendish square.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1829 †HEBERDEN, THOMAS, M.D., 98, Park street, Grosvenor square.

1849 HENRIQUES, AMOS, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley street, Portman square.

Elected

1821 Herberski, Vincent, M.D., Professor of Medicine in the University of Wilna.

1843 Hewett, Prescott Gardner, Surgeon-Extraordinary to H.M. the Queen; Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, Paris; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Trans.* 7. *Sci. Com.*

1855 Hewitt, Graily, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; Examiner in Midwifery at the University of London; 36, Berkeley square.

1872 Heyn, Julius Charles William, M.D., 88, Lange voorhout, the Hague, Holland.

1868 Hill, John Daniel, Surgeon to the Royal Free Hospital; Surgeon to the Royal Orthopædic Hospital; 17, Guilford street, Russell Square.

1862 Hill, M. Berkeley, M.B. Lond., Surgeon to University College Hospital, and Lecturer on Operative Surgery at University College, London; Surgeon for Out-patients to the Lock Hospital; 55, Wimpole street, Cavendish square.

1867 Hill, Samuel, M.D., 22, Mecklenburgh square.

1841 †Hilton, John, F,R.S., Surgeon-Extraordinary to H.M. the Queen; Consulting Surgeon to Guy's Hospital; Consulting Surgeon to the Royal General Dispensary, St. Pancras; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 4.

1868 Hinton, James, Aural Surgeon to Guy's Hospital; 18, Savile Row, Burlington gardens. *Trans.* 2.

1859 Hird, Francis, Surgeon to the Charing Cross Hospital; 13, Old Burlington street.

1861 *Hoffmeister, William Carter, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1872 Hogg, Francis Roberts, M.D., India.

Elected

1843 HOLDEN, LUTHER, Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865.

1814 †HOLLAND, SIR HENRY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; 72, Brook street, Grosvenor square. C. 1817, 1833-4. V.P. 1826, 1840. *Trans.* 1.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Physician to Casualty Department, St. Bartholomew's Hospital; 32, New Cavendish street, Cavendish square.

1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road south, Hampstead.

1856 HOLMES, TIMOTHY, *Librarian*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; Professor of Pathology and Surgery to the Royal College of Surgeons; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873. *Trans.* 5. *Sci. Com.*

1846 HOLT, BARNARD WIGHT, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.

1846 HOLTHOUSE, CARSTEN, Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; 3, George street, Hanover square. C. 1863.

1865 HOWARD, BENJAMIN, M.D., Lecturer on Operative Surgery, and Surgeon to the Long Island College Hospital, New York; 134, West 34th street, New York.

1865 HOWARD, EDWARD, M.D., Oaklands, Penge, Surrey.

1857 HULKE, JOHN WHITAKER, F.R.S., Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. *Trans.* 4. *Sci. Com.*

Elected

1857 HULME, EDWARD CHARLES, Woodbridge road, Guildford. *Trans.* 1.

1844 HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 5.

1866 HUNTER, CHARLES, 30, Wilton place, Belgrave square.

1873 HUNTER, WILLIAM GUYER, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-Major, Bombay Army, Bombay. [56, Great Cumberland place, Hyde park.]

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and to the Hospital for Diseases of the Skin; 4, Finsbury circus. C. 1870. *Trans.* 4. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.

1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., 1, Albert mansions, Victoria street, Pimlico.

1871 JACKSON, J. HUGHLINGS, M.D., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.

Elected

1868 JACKSON, THOMAS CARR, Surgeon to the Great Northern Hospital, and Surgeon to the National Orthopædic Hospital; 3, Weymouth street, Portland place.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.

1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.

1825 JAMES, JOHN B., M.D.

1839 JEFFREYS, JULIUS, F.R.S., 3, Broomfield, Bromley, Kent.

1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

1851 JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1864. *Trans.* 3.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Genevois."

1847 JOHNSON, GEORGE, M.D., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-4. V.P. 1870. *Trans.* 9.

1868 JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.

1848 JOHNSTONE, [JOHNSON], ATHOL ARCHIBALD WOOD, 20, Regency square, Brighton. *Trans.* 1.

1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.

1837 †JONES, THOMAS WILLIAM, M.D., 55, St. John's park, Upper Holloway. C. 1858.

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

Elected

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 16, Colmore row, Birmingham.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1872 KELLY, CHARLES, M.D., Assistant-Physician to King's College Hospital; 94, Wimpole street, Cavendish square.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1847 KEYSER, ALFRED, King's Hill, Berkhampstead.

1857 KIALLMARK, HENRY WALTER, 66, Princes square, Bayswater.

1851 KINGDON, JOHN ABERNETHY, *Vice-President*, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Trans.* 1. *Sci. Com.*

1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Trans.* 1.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; Consulting Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865.

1865 LANGTON, JOHN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 18, Harley street, Cavendish square.

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Paris.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

1862 LATHAM, PETER WALLWORK, M.A., M.B., Physician to Addenbrooke's Hospital, Cambridge; Deputy for Downing Professor of Medicine, Cambridge University; 17, Trumpington street, Cambridge.

Elected

1816 LAWRENCE, G. E.

1840 LAYCOCK, THOMAS, M.D., F.R.S.E., Physician-in-Ordinary to H.M. the Queen in Scotland, Professor of the Practice of Medicine and of Clinical Medicine, and Lecturer on Psychology and Mental Diseases in the University of Edinburgh; 13, Walker street, Edinburgh.

1843 *LEACH, JESSE, Moss Hall, Heywood, Lancashire.

1868 LEARED, ARTHUR, M.D., Senior Physician to the Great Northern Hospital; 12, Old Burlington street.

1822 LEDSAM, JOHN JOSEPH, M.D., 17, Esplanade, Scarborough, Yorkshire.

1843 LEE, HENRY, Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Trans.* 11. *Pro.* 1. *Sci. Com.*

1822 †LEE, ROBERT, M.D., F.R.S., Corresponding Member of the Academy of Medicine, Paris; 4, Savile row, Burlington gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 27.

1869 LEGG, JOHN WICKHAM, M.D., Physician to Casualty Department, St. Bartholomew's Hospital; 47, Green street, Park lane.

1836 LEIGHTON, FREDERICK, M.D., Frankfort-on-the-Maine.

1872 LIEBREICH, RICHARD, Ophthalmic Surgeon and Lecturer on Ophthalmic Surgery at St. Thomas's Hospital; 16, Albemarle street, Piccadilly.

1806 LIND, JOHN, M.D.

1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1824 †LOCOCK, SIR CHARLES, Bart., M.D., D.C.L., F.R.S., Member of the Senate of the University of London; 26, Hertford street, Mayfair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.

Elected

1846 LOMAX, HENRY THOMAS, Surgeon to the County Police; St. Mary's Grove, Stafford.

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen, Deputy Inspector-General, and Professor of Clinical and Military Surgery, Army Medical School, Royal Victoria Hospital, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.

1857 LYON, FELIX WILLIAM, M.D., 18, Buccleuch place, Edinburgh.

1867 MABERLY, GEORGE FREDERICK, Leamington, Warwickshire.

1873 MACCARTHY, JEREMIAH, M.D., Assistant-Surgeon to, and Lecturer on Physiology at, the London Hospital; 3, South street, Finsbury.

1867 MACCORMAC, WILLIAM, M.A., Surgeon to, and Lecturer on Practical Surgery at, St. Thomas's Hospital; 13, Harley street. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 1.

1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1866 MACGOWAN, ALEXANDER THORBURN, Kingswood park, near Bristol.

1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, and to the St. Anne's Society's Schools; Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 MACINTOSH, RICHARD, M.D.

Elected

1859 *M'Intyre, John, M.D., Odiham, Hants.

1873 McKellar, Alexander Oberlin, M.S.I., Resident Assistant-Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.

1854 *Mackinder, Draper, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1860 Maclean, John, M.D., 24, Portman street, Portman square.

1849 Maclure, Duncan Maclachlan, M.B., Lecturer on Physiology at the Westminster Hospital; Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 34, Harley Street, Cavendish square.

1842 Macnaught, John, M.D., 25, Bedford street, Liverpool.

1855 Marcet, William, M.D., F.R.S.; 1, Place Massena, Nice. C. 1871. *Trans.* 2. *Sci. Com.*

1867 Marsh, F. Howard, Assistant-Surgeon to St. Bartholomew's Hospital; 36, Bruton street, Berkeley square.

1838 Marsh, Thomas Parr, M.D.

1851 Marshall, John, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens, C. 1866. *Trans.* 2.

1841 †Martin, Sir James Ranald, C.B., F.R.S., Examining Medical Officer to the Secretary of State for India in Council; President of Medical Board for Examination of Officers of H.M.'s Indian Medical Service; Inspector General of Hospitals; 37, Upper Brook street, Grosvenor square. C. 1853. V.P. 1862.

1864 Mason, Francis, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. *Trans.* 1.

1869 Mayo, Charles, M.B., New College, Oxford.

1839 Meade, Richard Henry, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

Elected

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; Physician to the Hospital for Women, Soho square; 27, George street, Hanover square.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpellier row, Blackheath, Kent.

1867 MEREDYTH, COLOMIATI, M.D., 76, Margaret street, Cavendish square.

1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 25, Brook street, Grosvenor square.

1847 MERYON, EDWARD, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. V.P. 1868-9. *Trans.* 2.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1868 MICHELL, WILLIAM DANIEL, St. Peter's street, St. Alban's, Herts.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square. C. 1868.

1844 MONTEFIORE, NATHANIEL, 36, Hyde park gardens.

1836 MOORE, GEORGE, M.D., Priory Houses, Hastings, Sussex.

1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.B., Physician to the Manchester Royal Infirmary, and Lecturer on Medicine at the Manchester Royal School of Medicine; 1, St. Peter's square, Manchester.

Elected

1851 MOUAT, FREDERIC JOHN, M.D., late Surgeon-Major, Bengal Army; late Inspector-General of Gaols in the Lower Provinces of the Bengal Presidency, and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.

1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 6, Finsbury Circus. *Trans.* 1.

1856 MURCHISON, CHARLES, M.D., LL.D. Edinb., F.R.S., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital, Consulting Physician to the London Fever Hospital; 79, Wimpole street, Cavendish square. C. 1870-71. *Trans.* 3.

1873 MURRAY, IVOR, M.D., F.R.S. Ed., The Knowle, Brenchley, Kent.

1863 MYERS, ARTHUR B. R., Coldstream Guards' Hospital, Vincent square, Westminster; Windsor.

1859 NAYLER, GEORGE, Surgeon to the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park.

1843 NEWTON, EDWARD, 4, Upper Wimpole street. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Surgeon to the Brighton Children's Hospital; Surgeon to St. Mary's Hospital, Brighton; 11, Marlborough place, Brighton.

1849 NOVERRE, ARTHUR, 16, Park street, Grosvenor square. C. 1870-71.

1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

Elected

1870 NUNNELEY, FREDERICK BARHAM, M.D., 56, Friar Gate, Derby. *Trans.* 2.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1843 O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1858 OGLE, JOHN WILLIAM, M.D., Physician to, and Lecturer on Pathology at, St. George's Hospital; Inspector of Anatomy for the Provinces; 30, Cavendish square. C. 1873. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 98, Friar Gate, Derby.

1860 OGLE, WILLIAM, M.D., Lecturer on Physiology at St. George's Hospital; Oxford and Cambridge University Club. S. 1868-70. *Trans.* 4.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament Street].

1850 OLDHAM, HENRY, M.D., Consulting Obstetric Physician to Guy's Hospital; 4, Cavendish place, Cavendish square. C. 1865. *Trans.* 1.

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.

1873 ORD, WILLIAM MILLER, M.B., Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 11, Brook street, Hanover square.

1847 *PAGE, WILLIAM BOUSFIELD, Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., F.R.S., Sergeant-Surgeon Extraordinary to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-49. V.P. 1861. T. 1867. *Trans.* 9. *Sci. Com.*

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

Elected

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, House Surgeon to the Hospital for Sick Children; 49, Great Ormond street.

1841 PARKIN, JOHN, M.D., Rome.

1851 PART, JAMES, M.D., 89, Camden road, Camden town.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Physiology at, Guy's Hospital; 35, Grosvenor street.

1869 PAYNE, JOSEPH FRANK, M.B., Assistant-Physician to St. Thomas's Hospital; 6, Savile row, Burlington gardens.

1845 PEACOCK, THOMAS BEVILL, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. C. 1869. *Trans.* 2.

1856 PEIRCE, RICHARD KING, 16, Norland place, Notting hill.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 18, Temple row, Birmingham. *Trans.* 1.

1870 PERRIN, J. BESWICK, Medical Tutor, Royal School of Medicine; 10, Faulkner street, Manchester.

1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater.

1846 PHILP, FRANCIS RICHARD, M.D. [Colby House, Kensington.]

1867 PICK, THOMAS PICKERING, Assistant-Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 7, South Eaton place, Eaton square. *Sci. Com.*

1851 *PICKFORD, JAMES HOLLINS, M.D., M.R.I.A., 1, Cavendish place, Brighton.

1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1.

Elected

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.

1871 POORE, GEORGE VIVIAN, M.B., Assistant-Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street.

1843 POPE, CHARLES, M.D., Glastonbury, Somersetshire.

1846 POTTER, JEPHSON, M.D., F.L.S., Physician to the Liverpool General Hospital for Consumption and Diseases of the Chest; 6, Soho street, Liverpool.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 15, Henrietta street, Cavendish square.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; Examiner in Physiology at the University of London; 37A, Gt. Cumberland place, Hyde park. *Sci. Com.*

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Consulting Physician-Accoucheur to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. *Sci. Com.*

1869 PULLAR, ALFRED, M.D., Surgeon to the Kensington Dispensary; 47, Kensington park gardens.

1850 QUAIN, RICHARD, M.D., F.R.S., Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*

Elected

1835 †Quain, Richard, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 Radcliffe, Charles Bland, M.D., Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8.

1871 Ralfe, Charles Henry, M.D., M.A., Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.

1857 Ranke, Henry, M.D., Munich.

1854 Ransom, William Henry, M.D., F.R.S., Physician to the Nottingham General Hospital; the Pavement, Nottingham.

1869 Read, Thomas Laurence, 11, Petersham terrace, Queen's gate, Kensington.

1858 Reed, Frederick George, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 Reeder, Henry, M.D., Varick, Seneca County, New York, United States.

1857 Rees, George Owen, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Trans.* 1.

1869 Reeves, William, 5, the Crescent, Carlisle.

1855 Reynolds, John Russell, M.D., F.R.S., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; Examiner in Medicine at the University of London; 38, Grosvenor street. C. 1870.

1865 Rhodes, George Winter, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1847 Richards, Samuel, M.D., 36, Bedford square.

Elected

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., Warcop, Penrith.

1849 *RICHARDSON, WILLIAM, M.D.

1869 RICKARDS, WALTER, M.D., Physician to the Royal Free Hospital; 8, Cavendish place, Cavendish square.

1845 RIDGE, BENJAMIN, M.D., 21, Bruton street, Berkeley square.

1843 RIDGE, JOSEPH, M.D., 39, Dorset square. C. 1858. *Pro.* 1.

1863 RINGER, SYDNEY, M.D., Professor of Materia Medica in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square.

1871 *ROBERTS, DAVID LLOYD, M.D., Surgeon to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.

1852 ROBERTS, JOHN, M.R.C.P., the Park, Westow hill, Upper Norwood.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1843 ROBINSON, GEORGE, M.D. *Trans.* 2.

1843 RODEN, WILLIAM M.D., the Grange, Kidderminster, Worcestershire.

1829 ROOTS, WILLIAM SUDLOW, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Kingston, Surrey.

1850 ROPER, GEORGE, Bank House, Aylsham, Norfolk.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; High street, Hampstead. *Trans.* 1.

Elected

1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1834 RUMSEY, HENRY WYLDBORE, Priory House, Cheltenham.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital, and Professor of Medicine at Queen's College, Birmingham; 91, New Hall street, Birmingham.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S.E., Professor of Physiology at King's College, London, Fullerian Professor of Physiology to the Royal Institution; 12, Upper Berkeley street, Portman square.

1856 SALTER, S. JAMES A., F.R.S., F.L.S., Dental Surgeon to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. C. 1871. *Trans.* 2.

1849 SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3.

1855 SANDERSON, JOHN BURDON, M.D., F.R.S., Professor of Practical Physiology at University College; 49, Queen Anne street, Cavendish square. C. 1869-70. *Trans.* 2. *Sci. Com.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, Cheltenham.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician to the Royal Hospital for Diseases of the Chest, City road; 29, Duncan terrace, Islington. *Trans.* 1.

1845 SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

Elected

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital, 66, Brook street, Grosvenor square. C. 1871-2. *Trans.* 3. *Sci. Com.* 3.

1853 SCHULHOF, MAURICE, M.D., 46, Brook street, Grosvenor square.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1863 SEDGWICK, WILLIAM, Surgeon to the St. Marylebone Provident Dispensary; 12, Park place, Upper Baker street. *Trans.* 2.

1856 SERCOMBE, EDWIN, 41, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Professor of Anatomy and Physiology in University College, London; Member of the Senate of the University of London; University College, and Lawnbank, Hampstead. C. 1848-9. V.P. 1862.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.

1848 *SHEARMAN, EDWARD JAMES, M.D., F.R.S. Edin., F.L.S., Consulting Physician to the Rotherham Hospital; Moorgate, Rotherham, Yorkshire.

1859 SIBLEY, SEPTIMUS WILLIAM, 12, New Burlington street. *Trans.* 4. *Sci. Com.*

1849 SIBSON, FRANCIS, M.D., F.R.S., *Librarian*, Consulting Physician to St. Mary's Hospital; Member of the Senate of the University of London; 59, Brook street, Grosvenor square. C. 1863-4. L. 1873. *Trans.* 1. *Sci. Com.*

1848 SIEVEKING, EDWARD HENRY, M.D., *Vice-President*, Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873. *Trans.* 2. *Sci. Com.*

Elected

1871 SILVER, ALEXANDER, M.D., Physician to, and Lecturer on Physiology at, Charing Cross Hospital; 2, Stafford street, Bond street.

1842† SIMON, JOHN, D.C.L., F.R.S., Surgeon to St. Thomas's Hospital; Medical Officer of the Medical Department of the Local Government Board; 3, Parliament street, and 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.

1865 SIMS, J. MARION, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Nice.

1852 SMITH, CHARLES CASE, Consulting Surgeon to the Suffolk General Hospital.

1872 SMITH, GILBART, M.A., M.B., Visiting Physician to the Margaret Street Infirmary for Consumption, and Assistant-Physician to the Training Hospital, Tottenham; 68, Harley street, Cavendish square.

1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 2, Portugal street, Grosvenor square.

1835 SMITH, JOHN GREGORY, Medical Superintendent, Atkinson-Morley Convalescent Hospital, Copse Hill, Wimbledon, Surrey.

1843 SMITH, ROBERT WILLIAM, M.D., M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; Surgeon to Sir Patrick Dun's Hospital; 67, Eccles street, Dublin.

1838 †SMITH, SPENCER, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; 9, Queen Anne street, Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. *Trans.* 3. *Sci. Com.*

Elected

1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.

1845 SMITH, WILLIAM, 70, Pembroke road, Clifton, Bristol. *Trans.* 1.

1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1868 SOLLY, SAMUEL EDWIN, 11, Cleveland road, Barnes.

1865 SOUTHAM, GEORGE, Surgeon to the Manchester Royal Infirmary, and Lecturer on Surgery at the Manchester Royal School of Medicine; 10, Lever street, and Oakfield, Pendleton, Manchester. *Trans.* 4.

1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square.

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1851 SPITTA, ROBERT JOHN, M.B., Medical Officer to the Clapham General Dispensary; Clapham Common, Surrey. *Trans.* 1.

1843 *SPRANGER, STEPHEN, Cape Town, South Africa.

1867 SQUAREY, CHARLES EDWARD, M.B., Assistant-Physician to the Hospital for Women; 13, Upper Wimpole street. *Trans.* 2.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board. [Greenford House, Sutton, Surrey.]

1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.

1859 STEWART, WILLIAM EDWARD, 12, Weymouth street, Portland place.

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

Elected

1865 STOKES, WILLIAM, Jun., M.D., Professor of Surgery, Royal College of Surgeons, Ireland; Lecturer on Surgery at the Carmichael School of Medicine, and Surgeon to the Richmond Surgical Hospital; 3, Clare street, Merrion square, Dublin, *Trans.* 1.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square.

1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.

1863 STURGES, OCTAVIUS, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; 85, Wimpole street, Cavendish square.

1871 SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1869 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Assistant-Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health; Town Hall, Oldham.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 1.

1844 TAMPLIN, RICHARD WILLIAM, 33, Old Burlington street.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

Elected

1852 TAYLOR, ROBERT, Surgeon to the Central London Ophthalmic Hospital, and to the Cripples' Home, Marylebone road; 7, Lower Seymour Street, Portman square.

1845 TAYLOR, THOMAS, Warwick House, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1862 THOMPSON, EDMUND SYMES, M.D., *Secretary;* Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 3, Upper George street, Bryanston square. S. 1871-3. *Trans.* 1. *Sci. Com.*

1857 THOMPSON, HENRY, M.D., Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 4.

1862 THOMPSON, REGINALD EDWARD, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 21, South street, Park lane. *Trans.* 1. *Sci. Com.*

1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 60, Grosvenor street.

1872 TOMES, CHARLES S., B.A., Assistant-Surgeon to the Dental Hospital; 37, Cavendish square.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1828 TORRIE, JAMES, M.D.

1871 *TREND, THEOPHILUS W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.

1867 TROTTER, JOHN WILLIAM, Assistant-Surgeon, Coldstream Guards; Hospital, Vincent square, Westminster.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

Elected

1864 TUFNELL, THOMAS JOLLIFFE, Examiner in Surgery to the Royal College of Surgeons of Ireland; 58, Lower Mount street, Merrion square, Dublin.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1855 TULLOCH, JAMES STEWART, M.D., 1, Pembridge place, Bayswater.

1845 TURNER, THOMAS, F.L.S., Consulting Surgeon to the Manchester Royal Infirmary; 77, Mosley street, Manchester.

1870 VENNING, EDGCOMBE, Assistant-Surgeon, 1st Life Guards; Knightsbridge Barracks, and 24, Belgrave square.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital, and Ophthalmic Surgeon to the West London Hospital; 44A, Wimpole street, Cavendish square.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Hospital, Lisle street, Leicester square; 141, Regent street.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 25, Lowther street, Carlisle.

1867 *WALLIS, GEORGE, Benet street, Cambridge.

1852 WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.

Elected

1851 WALTON, HAYNES, Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.

1821 WARD, WILLIAM TILLEARD.

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Surgeon to the Metropolitan Convalescent Institution; Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, near Bristol.

1866 WARING, EDWARD JOHN, M.D. [Agents: Messrs. Grindlay and Co., 55, Parliament street]; 49, Clifton gardens, Maida vale.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Anatomy and Physiology in the Liverpool Royal Infirmary School of Medicine; 27, Hope street, Liverpool. *Trans.* 3.

1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals; 7, Henrietta street, Cavendish square.

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. *Trans.* 6.

Elected

1835 †WEBSTER, JOHN, M.D., F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 9, Queen street, St. Andrew's. C. 1843-4. V.P. 1855-6. *Trans.* 6. *Pro.* 1.

1844 WEGG, WILLIAM, M.D., *Treasurer*, Physician to the St. George's Dispensary; 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873.

1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row.

1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. *Trans.* 9. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2. *Sci. Com.*

1828 WHATLEY, JOHN, M.D.

1849 WHITE, JOHN.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1844 WILDBORE, FREDERIC, 245, Hackney road.

1870 *WILKIN, JOHN F., M.B. and C.M., Roxby House, Folkestone.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 77, Grosvenor street, Grosvenor square.

1863 WILLETT, ALFRED, Assistant-Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

Elected

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., *President*, Consulting Physician to the Hospital for Consumption, Brompton; 49, Upper Brook street, Grosvenor square. C. 1849-50. V.P. 1860-1. P. 1873. *Sci. Com.*

1859 *WILLIAMS, CHARLES, Assistant-Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 78, Park street, Grosvenor square. *Trans.* 2.

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square.

1859 WILLIAMS, JOSEPH, M.D. [3, Chichester street, Upper Westbourne terrace.]

1868 WILLIAMS, WILLIAM RHYS, M.D., Lecturer on Mental Diseases at St. Thomas's Hospital; Bethlehem Royal Hospital, Lambeth road.

1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.

1839 †WILSON, ERASMUS, F.R.S., Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. *Trans.* 2.

1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 WISE, THOMAS ALEXANDER, M.D., Rostellan Castle, Rostellan, County Cork.

1841 WOOD, GEORGE LEIGHTON, 27, Queen square, Bath.

1851 WOOD, JOHN, F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; Examiner in Anatomy at the University of London; Examiner in Anatomy and Physiology at the University of Cambridge; 68, Wimpole street. C. 1867-8. *Trans.* 3.

Elected

1872 WOOD, SAMUEL, St. Mary's Court, Shrewsbury.

1848 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8.

1833 †WORMALD, THOMAS, Consulting Surgeon to St. Bartholomew's Hospital; 42, Bedford row. C. 1839. V.P. 1854.

1842 WORTHINGTON, WILLIAM COLLINS, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.

1865 WOTTON, HENRY, Jun.; 62, Bedford gardens, Kensington.

1860 WYATT, JOHN, C.B., Surgeon-Major, Coldstream Guards; Hospital, Vincent square, Westminster.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., F.R.S., Cowley House, Oxford.

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France.

1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.

1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bickley, Kent.

1868 HOOKER, JOSEPH DALTON, M.D., D.C.L., LL.D., F.R.S., Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 26, Abbey place, St. John's wood.

1868 LYELL, SIR CHARLES, Bart., D.C.L., LL.D., F.R.S., Corresponding Member of the Academies of Sciences of Paris, Berlin, Philadelphia, Boston, &c.; 73, Harley street, Cavendish square.

1847 OWEN, RICHARD, D.C.L., LL.D., F.R.S., Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

Elected

1868 TYNDALL, JOHN, LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 ANDRAL, G., M.D., Member of the Institute and of the Academy of Medicine; Paris.

1872 BERNARD, CLAUDE, Member of the Institute of France, and of the Academy of Medicine; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Rue de Luxembourg, 24, Paris.

1862 CRUVEILHIER, JEAN, M.D., Physician to the "Hôpital de la Charité;" Member of the Academy of Medicine; Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden; President of the College of Health, and Director-General of Hospitals; Stockholm.

1841 EHRENBERG, CHRISTIAN GOTTFRIED, Foreign Associate of the Academy of Sciences of the Institute of France, Berlin.

1868 GROSS, SAMUEL D., M.D., F.C.P. Philad., D.C.L. Oxon., Professor of Surgery in the Jefferson Medical College of Philadelphia.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

1868 KÖLLIKER, ALBERT, Professor of Anatomy at Würzburg.

1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.

Elected

1868 LARREY, HIPPOLYTE BARON, Member of the Institute; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1850 ROKITANSKY, CARL, M.D., Curator of the Imperial Pathological Museum, and Professor of the University of Vienna. Referee for Medical and University Education to the Austrian Ministry; Vienna.

1856 STROMEYER, LOUIS, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

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REGULATIONS relative to the publication of the "Proceedings of the Society."

That, as a general rule, the Proceedings will be issued every two months, subject to variations dependent on the extent of matter to be printed.

That a Copy of the Proceedings will be sent, postage free, to every Fellow of the Society resident in the United Kingdom.

That "The Proceedings of the Society" may be obtained by non-members at the Society's House, 53, Berners Street, on pre-payment of an annual subscription of five shillings, which may be transmitted either by post-office order or in postage-stamps; —this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent, carriage free, through a bookseller, or by post, the receiver paying the foreign charges.

That a notice of every paper will appear in the Proceedings. Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the Proceedings *only*, or in the Proceedings and Transactions; and in all cases they will be expected to furnish an Abstract of the communication.

The Abstracts of the papers read will be furnished to the Journals as heretofore.

ON

NON-PURULENT CATARRH OF THE MIDDLE EAR.

BY

WILLIAM BARTLETT DALBY, F.R.C.S., M.B. CANTAB.,
AURAL SURGEON TO, AND LECTURER ON AURAL SURGERY AT,
ST. GEORGE'S HOSPITAL.

(Received August 3rd—Read October 8th, 1872.)

The object of this communication is to direct the attention of the Society to, 1stly, the several conditions under which the Eustachian tubes become so obstructed as to prevent the free passage of air from the pharynx into the tympanum; 2ndly, the various effects which follow catarrhal affections of the lining membrane of the tympanum, when the secretion is strictly of a mucous character, so far as they are observable during the life of the patient (this excludes all the effects of the purulent variety of catarrh of the middle ear); 3rdly, the treatment adapted for such cases.

The remarks which follow are based upon the notes of cases under treatment at St. George's Hospital during the last twelve months, and others under notice during the past three years. In the appendix to this paper only typical cases have been selected, and an abstract has been made of twenty of these.

The general term "obstruction of the Eustachian tube"

conveys but a very imperfect idea of the condition under which the patient who is the subject of it is suffering, and none whatever of the probable course of the affection, whether a few days or many months are likely to pass by before relief is obtained, or whether the obstruction is likely to be a permanent one. These differences are most markedly apparent in the case of children on the one hand, and adults on the other; in a less degree, but very distinctly, in individual cases of either class. In each it is sufficiently apparent that the impaired hearing which is always present, and the tinnitus which is occasionally so, are immediately in consequence of the regular supply of air in the middle ear being suspended, as both these symptoms disappear coincidently with an artificial inflation of the tympanum. In the case of adults, the Eustachian tube may become the seat of catarrh lasting for only a few days, giving rise to considerable deafness, which disappears without any treatment as the catarrh subsides; or the catarrh may entirely subside, leaving impaired hearing for an indefinite time. In some few cases this may be at once and permanently relieved by a single inflation of the middle ear on Politzer's method. In six of the cases this happened, no recurrence of the deafness taking place. In one the obstruction had lasted for six weeks, and in another for four. In none of these, after careful inspection was there any appearance either of the tympanum, or the faucial orifice of the Eustachian tube being affected; so it would seem that the obstruction must have been in some part of the course of the tube, (possibly due to a little mucus which was removed from its situation by the current of air); and that the mucous membrane lining the tube was not at the time secreting in excess; hence the non-recurrence of the deafness. More frequently the tube is the seat of catarrh lasting for some time, and it is then necessary to keep the middle ear inflated until it is recovered from. In those cases where the faucial orifice of the Eustachian tube was affected, astringent solutions were applied to the spot, apparently with considerable benefit. The longest period in adults during which it was found necessary to continue treatment was six

weeks, and as a rule, one tube only was affected at a time. In about one fourth of the cases, as air did not pass freely on Politzer's plan, the Eustachian catheter was employed, and on each occasion air could be heard through the otoscope to impinge on the tympanic membrane with a thud, and the patient was fully conscious of its so doing. From these results it would appear that there are two varieties of the affection in adults, the more chronic form showing in some measure a likeness to bronchitis. In both, the only abnormal appearances observed in the tympanic membrane were increased concavity, more than usual prominence in the short process of the malleus, and a tilting inward of the handle of this bone. The translucency of the membrane was unimpaired, and its natural curvature was resumed after a successful inflation.

In the case of children and young persons, the state of things is widely different, and may persist for months or even in severe instances for years. With them the whole mucous membrane lining the nares and fauces is affected, and the opening of the tube into the pharynx shares the same condition; their tonsils are enlarged, they breathe entirely through the mouth, and their well marked aspect is characteristic of this malady. In seven instances it was deemed advisable to remove the tonsils, as they interfered with the respiration and tended to keep up the unhealthy state of the throat.

In the most obstinate examples of this kind the good hearing which follows inflation of the tympanum will only last for a few hours, the patients afterwards becoming as deaf as ever. The perseverance in treatment which it may be necessary to exercise is well shown in case 2. The boy, fifteen years of age, had been very deaf for more than three years on both sides. The improved hearing after the first inflation lasted for only six hours. He was taught to use a Politzer's bag for himself, and for three months did this twice a day, thus keeping the hearing fairly good. After this, once a day for three months. The periods of good hearing increased in length, till in twelve months one inflation a week was only

necessary to maintain it. Astringent solutions were applied at various times to the throat. The case was under observation for eighteen months, at the end of which time, the boy had quite recovered.

Excepting in degree, this treatment in no way differs from that employed in all similar cases. It would seem that the desirable frequency for artificial inflation of the tympanum is proportionate to the length of time which improvements in hearing last, and that the seat of the obstruction with these patients is in chief part at the faucial opening of the Eustachian tube.

The next class of cases are those in which the mucous membrane lining the tympanum has become affected. This may either be in consequence of extension of the same condition from the Eustachian tube, or the cavity of the tympanum may become primarily attacked. In each instance the lining mucous membrane becomes swelled and secretes in excess. The first noticeable result of this is closure of the tube at its tympanic orifice, causing proportionate deafness. When these cases were seen a few days after the commencement of the attack, as the patients pressed air into the middle ear with the mouth and nose closed, the mucus in the tympanum could be detected by the moist squeaking râle which was heard through the otoscope. Only partial improvements followed immediately on using inflation. In some instances for the first few days, there was occasional pain in the ear, but never very severe, and in about half the cases more or less tinnitus.

The various abnormal appearances which the tympanic membrane exhibited at different stages of the affection were respectively as follows; one or more being present in all instances:—1stly, increase in the natural curvature; 2ndly, opacities; 3rdly, general thinning accompanied with a sodden appearance; 4thly, thinning in circumscribed spots; 5thly, perforation in these places; 6thly, calcareous degeneration.

A change from the natural to an increased curvature of the membrane which was observable in all the cases of not

long standing would be a natural consequence of the temporary closure of any part of the tube.

The various degrees of opacity which were invariably noticed would in like manner be easily explained, inasmuch as the innermost layer of the tympanic membrane was at the time a surface secreting unduly, no less than the other part of the mucous membrane lining the tympanum. It should be borne in mind, however, that opacities may be present whenever the external layer has suffered from changes taking place in the course of affections of the external auditory meatus; and therefore taken by itself, this appearance cannot be regarded as indicative either of previous or existing catarrh of the tympanum.

The general thinning of the membrane was only evident in those instances where the secretion was very copious and rapidly effused, and was frequently noticeable where the pressure from within the tympanum was eventually productive of a perforation.

It is especially important to direct attention to this change, as it is in these cases the question arises as to whether an attempt should be made to prevent a pathological opening (so to speak) in the membrane, by making an opening with a knife. If the first of these two issues occur, it cannot of course do so without more or less loss of substance taking place during the process of ulceration, and in a very large proportion of cases the perforation never heals; for very soon after the membrane has given way, the lining membrane of the tympanum begins to secrete purulent matter, and the same condition is arrived at which is seen when the perforation has taken place during the purulent variety of catarrh of the middle ear. In case 1, a small opening was made in the tympanic membrane with very good effect. The presence of fluid in the tympanum was amply evident by the sound which was produced upon passing air into this cavity, the patient herself being able to hear the fluid bubble. To judge from what happens under similar conditions, there would seem to be no reasonable doubt but that the tympanic membrane would have given way if no attempt had been made to relieve the

tension. The catarrh had lasted for twenty-eight days, the deafness was extreme, and only trifling and temporary benefit followed inflation with the catheter. How thinned the membrane had become was shown by the very slight resistance given to the knife as the incision was made; the state of the membrane might have been not inaptly described by comparing it to tissue paper soaked in water, altogether different from the crisp parchment-like consistence usually met with. On this as on other similar occasions, a small straight double-edged knife was employed, and light was reflected down a speculum from a reflector worn on the forehead. The hearing was completely regained, which certainly would not have been the case if the membrane had given way, or if the secretion had become inspissated in the tympanum.

The thinning of the membrane in circumscribed spots is not apparent by inspection alone, and it is necessary that the patient should inflate the tympanum while the membrane is being looked at. This appearance was more often observed some months or years after all symptoms of catarrh had passed away.

The precise shrunken aspect described by Dr. Adam Politzer, which led him to adopt the treatment of making an artificial opening and then to expel the mucus through the cut by passing a stream of air into the tympanum, was not seen in any of the cases; but it is fair to infer that it was a degree of the same condition behind the membrane which induced all the varied appearances that have been noticed.

The value which should be accorded to thickening of the tympanic membrane may be estimated by considering the effects of this change when the increase of substance is extreme. In a large number of the cases the tympanic membrane was the seat of calcareous deposit, and in several, the history would lead to the supposition that there had at some previous time been a perforation which had subsequently healed. Extensive, slight, and intermediate degrees of deafness were observable with this change, but against these must be placed a certain number in which calcareous deposits occupied large portions of the membrane, and this with per-

fectly good hearing on the affected side. It would seem probable from this, that the source of impaired hearing must be sought for in the changes which have taken place behind the tympanic membrane, and that the change in substance in this structure is not a serious matter excepting so far as being evidence of previous disease which may or may not have been recovered from.

A not very uncommon phenomenon when the mucus is in a fluid state in the tympanum is that great changes in the capacity for hearing may be induced by alterations in the attitudes assumed by the patients. Thus, in one case seen in June, 1870, after the catarrh had lasted for a month, as the patient lay with the head on the same level as the body, he could hear conversation quite well and a watch at six inches from the ear, and upon rising was in a few seconds extremely deaf to conversation and could hear the watch at only one inch. He recovered after six inflations of the tympanum with the catheter. In case 11 the patient was very deaf with the right ear, but on shaking the head violently and holding it to one side he could hear conversation very well, and a clock ticking at a distance of three feet, which before he could hear only when the ear was close to it. Injections of a warm solution of soda 5 grs. to the oz. were practised for ten times every other day. The ultimate result was good hearing.

In case 1 the order was reversed, as the patient who was hearing when she stood up became very deaf after lying down. In another case, a change from the erect to the prone position did not affect the hearing, but produced tinnitus. This symptom was relieved after an incision had been made in the membrane.

In these instances it would appear probable that a change in position of the secretion was the immediate cause of an alteration in hearing power.

Wherever there was obstruction within the tympanum on one side and a healthy ear on the other, the vibrations of a tuning-fork placed on the head were intensified in their effects on the deaf ear. This is readily explainable as the morbid products to the tympanum would interfere with the

outward passage of sound through the cavity, and so the vibrations would be reflected on to the labyrinth.

A very large proportion of the cases were not seen until long after the catarrhal condition had subsided, sometimes many months or even years. With these air passed into the tympanum with a dry sound, and no immediate improvement in hearing followed.

Where there were distinct nervous symptoms in addition to the tympanic disease no treatment was pursued; otherwise for the most part, it consisted in injections of warm solutions into the tympanum. Bicarbonate of soda 5 grs. to the oz. Hydrochlorate of Ammonia 4 grs. Iodide of Potassium 3 grs. Iodine vapour, or simply warm water. With some, a few drops of the solution was injected through the Eustachian catheter with the aid of an india-rubber bag. With others (the greater number) the following plan was adopted. The head was bent completely to the side to be acted upon, about half a drachm of the solution was put into the inferior naris of that side from a glass syringe, and as the patient swallowed it was forced into the tympanum with a Politzer's bag. This quite painless proceeding is especially useful for young children where considerable opposition is met with in using a catheter.

The general results of this treatment in cases apparently quite similar both in their history, and in the appearances of the tympanic membrane, are perplexing in the extreme. It seems to be almost impossible to say of any case seen for the first time, and examined ever so carefully, whether any benefit at all will result, and if so how much. Cases 12 and 20 were successful beyond all anticipations, but before and after these are seen to be case after case almost precisely similar in history and in none of them was there any appreciable improvement.

In case 20 a boy of nineteen years of age had a history of catarrh during childhood, was rejected for deafness by an army board, and could only hear a raised voice on either side. Injections of bicarbonate of soda, and afterwards of iodide of potassium were practised in all only six times, and the

middle ears were inflated on Politzer's method on the days after each injection, with the result of nearly perfect hearing on both sides. Before treatment the left ear was much the more deaf of the two, becoming afterwards rather the better one.

During life it is not surprising that it should be found to be quite impossible to estimate the changes which catarrh has wrought in the cavity of the tympanum. The amount of thickening of the lining membrane, situation of such thickening, the extent of immobility of the ossicles, the position occupied by the inspissated mucus—all these must be a matter of pure conjecture, and therefore arises the extreme difficulty of forming any opinion as to whether the injection of fluid into this cavity will lead to such softening of the dried mucus as to allow of its dislodgment by the air douche, for this is the manner in which this method of treatment is presumed to act. This presumption is favoured by the fact that in some cases, after injections have been practised for several successive days, on some single occasion upon inflating the tympanum, great improvement in hearing has followed, and has been permanent. In case 9 this was very noticeable. On a review of all the old standing cases the best results followed when the tympanum had been injected daily for about a week, and treatment resumed some weeks afterwards.

The number of cases where the operation was practised of making an incision into the tympanic membrane and afterwards passing fluid through the middle ear in the manner described by Mr. Hinton in the 'Guy's Hospital Reports' for 1869 was not sufficiently large to justify any conclusions being drawn as to the general value of this method of treatment in cases of tympanic disease of many years standing. The experience of the writer would lead him to limit this operation to those cases where there is unmistakeable evidence of the presence of mucus in a fluid or rather semi-fluid condition, as shown by the sound conveyed through the otoscope when the patient inflates the tympanum, as well as by the appearances in the membrane, and to place a further limit to this number by

restricting them to those instances where either the membrane was in danger of giving way from pressure within the tympanum, and to those others where more simple means have failed to induce the dispersion of the mucus, or at any rate its displacement from such situations as interfere with hearing. The number of such cases will necessarily be small, but where they can be distinctly defined there can be little doubt of the value of this treatment. In case 7 the patient himself could induce a bubbling in the tympanum each time he blew his nose. This was a source of great annoyance to him, but gave rise to very little deafness. After he had been treated by the air douche through the catheter for a time, and an interval of six months had elapsed, leaving him in the same condition, a small incision was made behind the handle of the malleus, the patient blew out a little mucus through the cut, the incision healed in two days and he had no more discomfort.

From the foregoing remarks it will be seen that in the cases of obstruction of the Eustachian tube when the tympana were not involved, the patients recovered, and the same may be said of catarrhal affections of the tympana which were seen in an early stage. On the other hand when the secretion had become inspissated before the patients applied for relief, when the sounds heard on inflation of the tympanum were of a dry character, the results were in a large proportion of the cases unsatisfactory, in others of these only partial improvements in hearing followed, and in a very few good hearing was effected.

In submitting these remarks to the Society the writer has attempted to make it clear that the result of careful observation has been to impress him with a strong conviction of the necessity of treatment in quite the early stage of the affection.

APPENDIX.

Containing an Abstract of Twenty Cases.

Subacute Catarrh of Tympana.

Case 1.—1870, May 30th, E. P—, girl, æt. 20. Five weeks ago could hear quite well. Then became deaf on left side, and a week later on the right; left tympanic membrane very opaque; right tympanic membrane translucent as in health; both drawn in more than natural. On passing air into the tympana with a Politzer's bag the hearing was slightly improved on both sides, but she was as deaf as before in an hour afterwards. Eustachian catheter was used for both ears every day for a week, the tympana being in this way vigorously inflated; considerably improved hearing at the end of this time, but still very deaf. The air passed into the tympana with a squeaking sound. On 20th June there had been a good deal of pain for two days in the left ear; inflation increased it. A small incision behind the malleus was made, and a little warm water passed through the tympanum and out at the nostril by means of a syringe made with a piece of india rubber on the nozzle which fitted the external meatus. On the 23rd the incision had healed and the hearing was good on that side. On the 18th, when the pain in the ear first began, she was hearing very well on this side; but after lying down on a sofa for a quarter of an hour she was quite deaf with that ear.

On the 25th June pain came as in the right ear, and by the 28th all the translucency was gone from the tympanic membrane, it had a sodden appearance; she was very deaf indeed with it, and on passing air into the tympanum there was a distinct bubbling sound. The same treatment was adopted for this ear as for the other and in making the incision there was very little resistance to the knife, probably due

to the fact that the membrane had become very much thinned. Some opaque fluid oozed through the cut which healed in forty-eight hours. The hearing in both ears now continued good until the early part of August, when there was a relapse on the left side. As this did not yield to ordinary measures the incision was repeated; good hearing following.

Last seen on September 15th, up to which date there had been no return of deafness.

Catarrh of Eustachian Tube.

CASE 2.—1870, September 7th, F. P—, æt. 15. Could hear well a little more than three years ago. Then became deaf, and in six weeks was nearly as deaf as now (in India at the time), but during the past six months has been getting rather worse; very deaf to conversation. Watch at two inches from the right ear, one inch from the left; mouth always open; does not breathe through nose; nares and fauces relaxed very much; no tinnitus. Both tympanic membranes quite translucent as in health, and the curvature increased to about an equal extent on each side. On passing air into the tympana with Politzer's bag the hearing was increased to three feet with a watch on either side, and was very fair for the voice. In six hours he was as deaf as before.

Treatment.—Occasional application of aqueous solution (ʒj to the oz.) of perchloride of iron to the pharynx. He was taught to use the bag himself, and did so as follows—twice a day for three months, once a day for next three months, every other day for next three months, twice a week for three months, and afterwards once a week.

In May, 1872, he heard well and had discontinued any treatment. No relapse occurred.

CASE 3.—1871, September 6th, M. D—, girl, æt. 15. Two years ago could hear quite well; at that time she became deaf during a cold, and has been getting slightly worse ever

since. Both tympanic membranes are very opaque. The hearing on the right side is fairly good for conversation addressed immediately to her by a person sitting near, and the watch is heard at thirteen inches from the ear. On the left side she is very deaf indeed, and can hear the watch at one and a half inch from the ear. Tuning-fork heard well when placed on the vertex. No nervous symptoms. Politzer's inflation gave no improvement, the air impinging on the tympanic membrane with a dry sound. Soda solution was injected into this as well as the other tympanum four times. No improvement followed. This treatment was resumed again in three weeks but with same results.

Case 4.—1871, September 19th, J. E—, æt. 46. On the 12th August went to sleep close to open window, the door of room being also open at the time. Slept for two hours, and then went to bed. The following morning was very deaf in left ear, and slightly so with the right. Since then has been getting worse with the left side. Now very deaf to conversation and hears a loudly ticking watch only when in close contact with the ear (left). The tympanic membrane very opaque. Moist sound accompanies inflation of the tympanum. After two inflations the right side recovered (the translucency of the tympanic membrane was not at all impaired), but only temporary improvements were effected on the left side. The Eustachian catheter was used every day for six times, three times for injection of soda solution, 5 grs. to the oz., and three times for air douche. The hearing was now much better, and on October 3rd he reported that he heard well. There was no relapse.

Case 5.—1871, September 29th, M. V—, æt. 20. Attended with a polypus and perforation of the right tympanic membrane. During treatment the left ear became affected in following manner:—After not coming to the hospital for a week, on the next visit she said that five days previously she became slightly deaf on the left side. This had increased very much, and occasionally there had been pain in the ear

lasting for two or three hours. The tympanic membrane had lost its translucency; air entered the tympanum with rather a moist sound, improving the hearing for a few hours afterwards. A week later she came to hospital saying that the hearing had become worse until the previous day, when upon waking in the morning there was a little oozing from the ear and she heard much better. A slight perforation could be seen behind the handle of the malleus.

The perforation had healed by the next week, but seven days afterwards the membrane gave way in the same place. A discharge from the ear followed and in July, 1872, the perforation had not healed. The hearing though impaired was not very much so.

Case 6.—1871, December 20th, M. C—, delicate-looking girl, æt. 15, never heard at all with right ear. Two years ago attack of deafness on left side, which passed off in two days. Twelve months ago hearing became very imperfect on left side and has been getting worse. Watch is heard at two inches from the ear and conversation very badly indeed. The tympanic membrane somewhat retracted, and rather opaque. Tuning-fork heard fairly on the vertex. Inflation on Politzer's method gave some slight improvement but very little. Not seen again until April 10th, when the hearing was considerably worse than on former visits.

Treatment.—Injection of solution of iodide of potassium every other day, and inflation daily, for fourteen days. The hearing gradually improved until she could hear conversation in an ordinary tone from a person sitting close to her. This state of hearing, which was a great advance on the condition before treatment, was maintained. In the month of March of the same year she had been taught to speak on the fingers as the difficulty of holding conversation was so great. Injections were resumed again in June for a week, but no further improvement followed their use.

Chronic Catarrh of Tympanum.

Case 7.—1871, December 21st, J. C—, æt 30. Four years

ago well. Then some affection of the throat lasting a few weeks. A stuffed feeling in left ear at intervals ever since. This sensation has been more troublesome during the last six weeks. The hearing is perceptibly, but not much impaired. The tympanic membrane is opaque, and the outline of the handle of the malleus is indistinct. As he passes air into this tympanum a squeaking bubbling sound is very distinctly audible through the otoscope, or if listened for close to the ear without the otoscope.

Treatment.—The catheter used four times on successive days with some benefit. He then went home to Manchester and returned on May 27th, 1872, no better than on the first visit. Then a small opening was made; he blew out through the cut a very little mucus; the incision had healed on the 29th. Dismissed on June 1st well, and had no recurrence of the unpleasant symptom. Before the incision was made, the vibrations of a tuning-fork placed on the vertex were heard much louder on the deaf side. Afterwards there was no perceptible difference for either ear.

Case 8.—1871, December 28th, E. G—, girl, æt. 8. Very deaf for four years with both ears, especially with the right, speech impaired in consequence, breathes entirely through mouth which she keeps always open. Both tonsils enlarged, but the right very much so. The right tonsil was removed, inflation of the tympana on Politzer's method twice a week until February 9th, when she was discharged hearing as well as she had done during the previous fortnight.

Case 9.—1872, January 14th, A. W—, æt. 26. At Christmas became deaf in right ear, having a cold at the time. Now extremely deaf to conversation, and a watch heard at four inches which can be heard at twelve feet with the other ear. Tympanic membrane opaque, and malleus retracted. Inflation on Politzer's method improves the hearing very slightly, but the change only lasts for an hour. Injections through the Eustachian catheter were practised six times altogether, with the result of very little change in hearing

power until the last occasion when the hearing immediately afterwards became natural, and there was no return of the deafness subsequently.

CASE 10.—1872, January 24th, J. W—, æt. 18. Two months ago he could hear well. Then became deaf in left ear, and a week afterwards in the right. Both tympanic membranes of natural translucency, curvature increased. Inflation with Politzer's bag gives some improvement, but not very much. On the right side excellent hearing followed the air douche through the catheter, and after this had been repeated three times he could hear well on this side. On the left side, however, the catheter would not pass through the inferior naris as the vomer was bent very much to this side, giving a crooked shape to the nose. It was not until after several attempts that air passed freely into this tympanum on Politzer's method; when it did so the hearing improved, but not permanently until after the inflation had been practised once a week for eight weeks.

Subacute Catarrh of Tympanum.

CASE 11.—1872, January 30th, F. F—, æt. 50. Ten years ago could hear quite well, then became slightly deaf and remained so for twelve months, but recovered without treatment. On four separate occasions has had attacks of deafness which have been recovered from on proceeding from England to Spain and remaining a few weeks in the latter country. Three weeks ago was hearing as usual and became deaf in right ear gradually. This, the better ear of the two generally, is now the worse. Conversation only heard with a raised voice, and a watch when pressed firmly against the ear; throat somewhat relaxed; the curvature of the membrane on the right side considerably increased; translucency not very much impaired; vibrations of tuning-fork on head intensified on this side. On shaking the head and bending it downwards can hear fairly well.

Treatment.—Inflation for three successive days, giving some

slight, but only transient improvement. Not seen again till March 9th, no better. Every other day for ten times solution of carbonate of soda, 5 grs. to the oz. through the catheter. On April 10th, hearing quite good on this side.

CASE 12.—1872, February 17th, J. H—, æt. 35. At ten years of age measles, occasional pain at the time in the right ear, followed by discharge which remained for a few weeks and then stopped. After this, deaf on this side as now. Five years ago pain on several occasions in left ear, this passed off leaving good hearing; never any discharge on this side. Three months ago became deaf with this ear and has remained so since. At present very deaf to conversation with both ears, but hears somewhat better on the left side. The tickings of watch heard at one inch from each ear. Occasionally slight tinnitus on left side. Right tympanic membrane very much fallen in and opaque. Left tympanic membrane opaque; not any very perceptible change from natural curvature. Air entered both tympana on Politzer's plan.

Treatment.—Injections of carbonate of soda, 5 grs. to the oz., into both tympana for seven successive days. Afterwards every third day till March 6th. Hearing on left side gradually improved, and some improvement though not very much on the right side. On this date he could hear as well as ever on the left side. Dismissed. Seen in July and reported there had been no change.

CASE 13.—1872, April 17th, L. H. F—, æt. 5. Parents noticed two years ago that he was slightly deaf and he has continued so ever since. The last three months when on voyage from Australia been getting worse, and his speech in consequence is becoming impaired. Both tympanic membranes more concave than natural; translucency unimpaired; rather enlarged tonsils. Watch heard only when in contact with both ears. Requires a raised voice rather close to hear what is said to him. Inflation on Politzer's method gave good hearing which lasted on the first occasion for forty-eight

hours. Solution of perchloride of iron applied to the throat. The tympana inflated every day for six days. Then twice a week for two weeks. Then once a week until July 1st. Dismissed hearing well and has had no return of deafness.

Case 14.—1872, May 4th, M. S—, could hear quite well two weeks ago when she became deaf in both ears. After four or five days felt a crack in right ear, which was followed by good hearing and has since had no return of deafness on that side. Some tinnitus and very bad hearing on the left side. This was completely relieved by one inflation on Politzer's method, and no further trouble. The tympanic membrane was healthy with the exception of some increase in the curvature. No relapse.

Case 15.—1872, March 19th, M. H—, girl, æt. 20. In 1863 could hear well, then became deaf from a cold and completely recovered without treatment in about a week. From that time has been subject to attacks of deafness; each fresh attack leaving the hearing more impaired than the last one; the right tympanic membrane translucent; the left opaque; much more deaf with a cold; no nervous symptoms or tinnitus; tuning-fork placed on vertex heard well. Inflation of tympana gave no improvement. Injections of soda, 5 grs. to the oz., for four days in succession. After the lapse of a week for three days in succession, no perceptible change in the hearing of either side.

Case 16.—1872, May 9th, E. L—, girl, æt. 19. Has been very deaf with both ears for the last two years, requires raised voice near the ear to hear conversation. Tympanic membranes both translucent, but very much more concave than natural. Air entered both tympana on Politzer's method with a bang increasing hearing from one inch with a watch from the ear to three feet. Both tonsils very much enlarged, affecting voice and breathing. Tonsils both removed with guillotine. Tympana inflated twice a week; on each occasion the periods of good hearing increased in length.

On June 27th, a fortnight had elapsed and good hearing had been maintained during that time. Discharged.

Case 17.—1872, May 22nd, B. H—, æt. 23, delicate looking girl, could hear quite well a week ago, became in a few hours very deaf in right ear. Previously had relaxed throat which was recovered from. At present—left ear, good hearing and healthy membrana tympani. On using Politzer's inflation on the first occasion air did not pass into the right tympanum. On the second occasion it did so, increasing the hearing from six inches with a watch to two feet. The tympanic membrane on this side was very much more concave than natural, and the translucency was impaired though not to any very great extent. The improvement from inflation only lasted for four hours. Vibrations of a tuning-fork placed on the vertex were much intensified on this side.

Treatment.—Injections of carbonate of soda, 5 grs. to the oz., for four successive days. Inflation on two separate visits at an interval of three days. Hearing perfect on 29th May. No recurrence of deafness.

Case 18.—1872, June 10th, E. M—, girl, æt. 5. Six weeks ago could hear well, now very deaf to conversation, a watch at one inch from the right ear, and three inches from the left. Relaxed throat, both Eustachian tubes obstructed, good hearing immediately on both sides after using Politzer's inflation, became deaf again in eight hours though not so much as before. Perchloride of iron solution was applied every day for eight days to the throat. Politzer's bag used every day for two weeks, afterwards twice a week for three weeks, once a week for three weeks. Then hearing good, and no recurrence of deafness.

Case 19.—1872, June 13th, C. E—, æt. 30. One week ago she could hear well. Now watch at one inch from the right, and in contact with left. Translucency of both membranes unimpaired, air entered (with a bang) both tympana, giving natural hearing. No recurrence of deafness.

Case 20.—1872, June 19th, R. W—, æt. 19. Never heard well with the left ear, and now depends chiefly on the right. Scarlet fever when a child, but no history of discharge from either ear. Very deaf to conversation. Refused commission by an Army Board on the ground of deafness last year. Watch heard one inch from the right ear and half inch from the left. Vibrations of tuning-fork on vertex heard well. Both tympanic membranes very opaque, and concavity increased. Inflation on Politzer's method gave slight improvement, but lasting for a few hours only.

Treatment.—Injection of solution of bicarbonate of soda, 5 grs. to the oz., for five days successively. Vigorous inflation for three days; injection of iodide of potassium, 4 grs. to the oz., for four days, followed by vigorous inflation for two days. Dismissed on July 3rd, conversation heard perfectly well with the left ear (this was originally the worse of the two) and very nearly good hearing with the right. To use inflation himself occasionally during next two weeks. No relapse occurred.

CASE

OF

OVARIAN DROPSY,

OPERATED ON DURING AN ATTACK OF ACUTE PERITONITIS.

BY

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COMMUNICATED BY

T. SPENCER WELLS, F.R.C.S.

(Received August 5th—Read October 8th, 1872.)

THE operation of ovariotomy has been so frequently and successfully performed by so many, and in all parts of the world, that it is no longer necessary to publish cases for the purpose of proving the legitimacy of the operation. Results have established its position as being one of the most successful and life-saving of all the capital operations in surgery.

It must, however, be forcibly impressed on any one who has extensive opportunities of observing disease of the ovaries that there are scarcely two cases which come under operation presenting exactly similar features; varieties in the tumour and its contents, in its adhesions and relations

to the organs of the abdomen or pelvis, in the length, thickness, or vascularity of the pedicle, all require attention, and useful lessons are to be learned from nearly every case. Hence the mistake often made by onlookers at those operations; perhaps a surgeon who has never performed ovariotomy is present at an operation on a unilocular cyst uncomplicated by adhesions, with a pedicle of good length, he remarks, "What a simple operation! nothing in it;" let him, however, watch a dozen cases, and he will find that some of them require as much boldness, dexterity, and readiness of invention as any other operation in surgery.

I have had as good opportunities for seeing and treating cases of this class as can be afforded to any practitioner in this colony of Victoria, having been connected with the Hospital for Women in Melbourne since 1856, but in a young country like this with a healthy population the cases of ovarian disease are few indeed compared with those which come under notice in the large cities at home.

I have now operated seventeen times. Of those, thirteen have recovered and are at the present time alive and in good health, four have borne children since the operation; in two of the cases that died the tumour proved to be malignant. I have had in this small number of patients nearly every variety of complication described by the most experienced operators and have reported them from time to time in the 'Australian Medical Journal;' but the case which I now wish to bring under the notice of the members of the Royal Medical and Chirurgical Society of London seems to me worthy of being so placed on record, and though I am quite aware similar cases have been reported by Mr. Spencer Wells and others, still I feel assured that no case has ever been operated on in which the prognosis could have been worse, and I almost fear that I shall be suspected of exaggeration when I give only a plain statement of facts.

I first saw the patient, Mrs. G—, on the 30th of April, 1872. She had been brought a journey of twenty miles by railway the day before. At this interview I ascertained the following history:—Thirty-one years of age; married five

years; has had two children, the last two and a half years ago.

She is very much emaciated, breathing with great difficulty on account of the enormous distension of the abdomen; has frequent attacks of hectic and great irritability of stomach; pulse 110, very feeble. Her distress was so great that I felt compelled to give her temporary relief by tapping, before her removal to the hospital.

On March 1st (next day) my colleague, Dr. Martin, saw her in consultation, and assisted me in tapping one portion of the tumour, which was evidently multilocular and in portions semi-solid. I introduced the trocar into the most fluctuating portion and drew off two quarts of thick muco-colloid fluid; this emptied one cyst, and gave some little relief.

The place in which she was staying being most unsuitable she was carefully removed on the 5th of March to the hospital. I should have stated that the whole surface of the abdomen was extremely tender to the touch. After admission to hospital the history of the case was more carefully taken. She stated that she first noticed an enlargement in the left iliac region about fifteen months ago; this went on gradually increasing, and after a time she believed herself to be pregnant, and even fancied that she felt the movements of the child. Her health got worse and worse, but she did not seek advice, believing all her sufferings to be due to pregnancy. Some time before coming to Melbourne she had, however, consulted a medical man who at once told her she was not pregnant and sent her to see me. When admitted she measured thirty-eight inches round the abdomen at the umbilicus, nine inches from ensiform cartilage to umbilicus, and eight and a half inches from umbilicus to pubis.

For many days after admission she was very weak, and was unable to retain food on her stomach; she was supported by enemata of beef tea and brandy, had warm fomentations and linseed-meal poultices to the abdomen, small doses of opium, and effervescing draughts, and was anxiously watched in the

hope that she would gradually improve sufficiently to have the operation performed. For some days she appeared to improve a little, but about the 16th of March she became much worse, and it was evident that she would soon sink. The temperature of her body rose rapidly, 102° in axilla, the vomiting increased, the tenderness over the abdomen became greater; pulse 120°, small.

After consultation with my colleague I determined to operate as affording the only possible chance, and was much encouraged in this resolve by the perusal of a somewhat similar case reported by Mr. Spencer Wells in the 'Medical Times and Gazette.'

The operation was performed on the 19th of March in the presence of about a dozen medical men and my class of students from the university. Bichloride of methylene was administered by Dr. Motherwell, and I was ably assisted by my colleagues, Drs. Martin and Featherstone and Dr. James. When the patient was placed on the table the pulse was almost imperceptible, the body covered with a clammy sweat; temperature in axilla 102°. A long incision was made extending about an inch above the umbilicus. On opening the cavity of the peritoneum several pints of ascitic fluid mixed with flakes of lymph escaped; on introducing the hand, adhesions were found all over every aspect of the tumour. These were recent and easily broken down by the hand. On inserting the large syphon trocar the contents were found to be too thick to flow through the tube. I opened the tumour freely and introduced my hand breaking down cyst after cyst, giving exit to several different kinds of fluid—thick muco-colloid fluid, purulent, and the ordinary glairy fluid of ovarian disease; the walls of several cysts were too tough to break down and required puncture with the smaller trocar. When sufficiently reduced in size the mass was withdrawn from the abdomen. The pedicle was very broad but of a good length and was secured simply by a clamp. The entire cavity of the abdomen was of a deep purple or maroon colour; the parietal layer of peritoneum, when cut by the primary incision, was as thick as wash-leather, and did not

retract in the least. The intestines were of the same deep purple colour and were matted together with lymph. They were closely packed away in the upper part of the abdomen to the right side. Enormous quantities of soft shreddy lymph were found adherent to various parts of the peritoneal surface, particularly to the under surface of the diaphragm, and about the crura of that muscle it was in such quantity that I removed it in handsful. Assiduous sponging at length removed the effused lymph from all parts as perfectly as was possible. The wound was closed by eight deep silk and six superficial horsehair sutures, the pedicle trimmed and dressed with a powder composed of gypsum and carbolic acid, broad strapping of adhesive plaster and a flannel roller applied, and the patient removed to bed. The operation occupied thirty-six minutes.

Nothing could be better than the effect of the chloro-methylene; it caused no dyspnœa or sickness and the patient recovered perfect consciousness almost immediately after being placed in bed. A suppository of ten grains soap and opium pill was introduced, and she took some iced water with a little brandy.

At 5 p.m., two hours after operation, pulse was 124; temperature 102°. At 10 p.m. she had slept a good deal; no sickness; pulse 120; temperature down to 96°; said she felt very comfortable.

The solid portions of tumour weighed 11½ lbs., and the fluid removed (not including the ascitic fluid) measured 22 pints.

Next day she was doing perfectly well, passed urine herself, took some tea and toast.

On the second day she had a slight motion and passed a great deal of flatus.

At 10 p.m. on the 21st, second day, six of the deep sutures were removed; wound healing well. Enemata composed of two ounces of very strong beef tea and one ounce of brandy were given three or four times a day for the first week.

On the third day she had some chicken for dinner; has had no sickness of stomach since the operation.

On the fourth day she was not so well; very restless; and said she felt worse than at any time since the operation.

When dressing the pedicle I moved the clamp in order to cleanse the root of the pedicle and a quantity of most fœtid gas escaped from the lower part of the wound, followed by about eight ounces of fearfully fœtid, thin, greenish-coloured fluid. The parts were carefully sponged with permanganate of potash solution in tepid water and dressed with chloralum wool. All sutures removed; wound healed well down to clamp. She never had a bad symptom afterwards. The clamp came away on the eighth day, and a little more fluid then escaped from the abdomen; after that the wound closed rapidly. She gained strength daily, was sitting up on the twelfth day, and left the hospital perfectly well on the 15th of April.

It is unnecessary for me to dilate on the many points of interest in this case; one or two, however, seem to me important. I have no doubt whatever that the bichloride of methylene is infinitely superior to chloroform in these cases, it causes no sickness or depression, and the patient is much sooner free from the anæsthetic influence.

In every case that I have done as yet I adhere to Mr. Spencer Wells's mode of treating the pedicle by the extra-peritoneal method, and I feel sure that this patient would have been in a very critical position if the pedicle had been dropped in and the wound closed, thereby preventing the evacuation of the fœtid fluid which formed as a result of the engorged state of the peritoneum. Of course, I would not wish it to be understood that I would hesitate to drop in the pedicle in a case where it was very short and would be strained by a clamp; but, as far as my small experience goes, I would bring out and clamp every pedicle that was sufficiently long to be so treated.

ON THE

MORBID EFFECTS OF ALCOHOL,

AS SHOWN IN

PERSONS WHO TRADE IN LIQUOR.

BY

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In my work on 'Albuminuria'* I brought forward facts which appeared to indicate that the injurious effect of alcohol upon the kidney had been exaggerated. My conclusions in their turn have been disputed. But it is not now my purpose to pursue the subject controversially, but to lay before the Society some details which have been extracted from the records of St. George's Hospital, the searching of which I began with especial relation to the glands in question, but have pursued with a widened scope, having regard to the effects of alcohol upon the whole body rather than upon any organ in particular.

The character of the books I have referred to is here well known. They are the work of a line of competent pathologists; they are systematic and complete, and insomuch as the observations were made without special object, we may believe them to be generally unbiassed.

I have started with the assumption that a person in whose

* Chapter XVI. "Alcohol as a Cause of Renal Disease."

vocation alcoholic drink, to be had for nothing, is ever suggestively present, will on an average take more of it than one in whose calling such liquor plays neither a necessary nor a gratuitous part. It needs but a slight experience of human nature to justify this surmise. The mouth is not muzzled of the ox who treadeth the corn or of the potman who carries the beer. With a reliance upon this general truth, justified by the notorious inebriety and the liability to delirium tremens of those who trade in drink, I have contrasted the morbid appearances in the bodies of people of this class with those in the persons of others who have neither been professionally conversant with intoxicating liquids nor known to have been addicted to their intemperate or excessive use.

It is scarcely necessary to observe that any pathological observations as to the effect of alcohol to be valid must be comparative. Bare statements that among so many dead drunkards many or few were tuberculous, while in such a number the liver was cirrhosed or the kidneys granular, convey little information. It is necessary to know how frequent such lesions are in persons similarly circumstanced in other respects save drink. I have, therefore, used a strictly comparative method, and have applied it to as large a number of dissections, and to as many particulars in each, as the materials permitted.

The post-mortem books of St. George's Hospital, which were begun with the year 1842, contain in chronological order a detailed account of every examination made from that date to the present time. Subsequent to the beginning of 1844 the history and clinical particulars corresponding to each are adjoined. From the commencement of 1842 to the end of 1871, a term of thirty years, I have abstracted the morbid appearances, and, with the exception of the first two years, the leading clinical facts relating to every person examined after death whose trade it had been to make, sell, store, or convey alcoholic liquor. As a counterpoise or standard of comparison for each such case (not that it was designed to compare single cases one with another, but in order to obtain two series as similar as possible, save in respect

of drink) I took after each alcoholic post-mortem one upon a person of the same sex, not employed about liquor nor known to have had delirium tremens or to have been of intemperate habits. In order to avoid bias in selection I went by rule. After each alcoholic examination I abstracted the non-alcoholic next afterwards, except where the end of the annual volume compelled me to turn backwards instead of forwards. Since the alcoholic series began at the age of sixteen, all post-mortems upon younger subjects were excluded. Also were excluded those which referred to workers in lead, the effect of this metal upon the kidneys possibly tending so far to obscure the result.

Thus, were contrasted two series of post-mortem examinations the subjects of which had been differently placed with regard to drink, but in no other respect were obviously dissimilar. They were of the same sex, of similar age, they belonged to the same stratum of society, they had been patients at the same time in the same hospital, and were at last examined, and the results recorded, by the same observers.

Each series comprised 149 post-mortem examinations*—146 men, 3 women.

The traders in liquor were thus divided:

Potmen	54
Waiters	43
Cellarmen	11
Draymen	11
Brewers or brewers' men	9
Barmen	8
Publicans	8
Barmaids	3
Distillerymen	1
Maltsters	1
	149

The opposed class, that not concerned with alcohol, com-

* Full details of every case, in a tabular form, were laid before the Society. These tables, which were very voluminous, formed the basis of the abstracts which are printed.

prised, as will be seen below, a great variety of occupations. The lead trades were excluded for reasons which have been stated; while billiard-markers, butlers, wine-coopers, and excisemen were not admitted on either side, their vocations holding with regard to drink an uncertain attitude.

In the course of the enquiry, the questions will arise how far certain differences in the morbid proclivities of the two classes are to be attributed to causes other than alcohol. Chiefly how far do the classes differ in exposure to weather, one undoubted agent in the production of rheumatic and inflammatory disease.

The non-alcoholic traders are therefore arranged so as to show as nearly as may be how many work in the open air, how many in houses, and with how many their place of labour is uncertain or variable.

Occupations of the non-alcoholic traders, arranged according to their probable exposure to weather.

Indoor.		Mixed or uncertain.		Outdoor.	
Footmen or pages ...	6	Grooms	8	Labourers	21
Clerks	4	Railway or other porters	6	Coachmen, carmen, or postillions......	16
Smiths or metal turners	4	Carpenters	5	Gardeners	5
Tailors	4	Stablemen or horse-keepers	4	Travellers or collectors	4
Shoemakers	4	Old soldiers or pensioners	4	Commissionaires or errand boys	3
Schoolmistress or wife	3	Plasterers	3	Sailors	3
Bakers	2	Butchers	3	Watermen or barge-men	2
Upholsterers	2	Stonemasons	3	Milkmen	2
Greengrocers	2	Watchman, detective, sweep, engineer, shipwright, woodcutter, chaff-cutter, veterinary surgeon, of no occupation, of each 1	9	Hawker, brickmaker, postman, of each 1	3
Cooper, wheelwright, bird-stuffer, ironmonger, mattrass maker, dentist, merchant, hair-dresser, draper, tallow-chandler, saddler, cabinet-maker, fishmonger, brush-maker, of each 1	14				
Total	45	Total	45	Total	59

Hence it appears that, excluding the callings of uncertain or mixed character the outdoor trades preponderate as 59 to 45, or about 4 to 3.

With the traders in liquor indoor pursuits are the more numerous. Waiters, cellarmen, brewers, publicans, barmen, barmaids, distillers, and maltsters, all generally under cover, together amounted to 84; potmen and draymen, mostly in the open air, together came to 65. Thus, the ratio of 4 to 3 will nearly represent both the preponderance of indoor occupations with the alcoholic series, and of outdoor occupations with the non-alcoholic.

Comparing the two classes, the alcoholic and the non-alcoholic, the results are these:

Age.

The alcoholic traders who die in the hospital attain a mean age of 36·8; the non-alcoholic a mean age of 40·6.

Excluding all deaths from accident, dealing solely, therefore, with the effects of disease, the figures are practically unaltered; alcoholic traders dying at the age of 36·8, non-alcoholic at the age of 39·9.

It, therefore, may be stated with general truth that to deal in liquor costs 3½ years of life.

The shortness of life in both classes is, no doubt, owing to the fact that a hospital is essentially the resort of disease not of age. A workhouse would have given different results, though, possibly, the proportion would not have been much altered.

The following statement of the number of deaths in each decade of life in the two series shows that the mortality from alcohol is most marked between the ages of 30 and 40.*

* The increased mortality in the fourth decade among the alcoholic was chiefly due to delirium tremens, phthisis, diseases of the liver and kidneys, and sanguineous apoplexy. I have given some further details in the 'British Medical Journal' for 1873, p. 8.

Age at death.	Alcoholic trade.		Non-alcoholic trade.
From 16—20	10	...	11
21—30	37	...	39
31—40	50	...	28
41—50	31	...	32
51—60	12	...	21
61—70	5	...	13
71—80	1	...	4
81—90	0	...	1

I will now consider the morbid changes pertaining relatively to the two classes, necessarily confining the inquiry to the more obvious and tangible, and taking the organs in the order in which they would be reached by a fluid absorbed by the veins of the stomach.

This course is sufficiently obvious. Anything which enters by the veins of the stomach and survives admixture with the blood must needs first pass through the liver. Leaving it by the hepatic vein it enters the right side of the heart and is there mixed with other venous streams. Thence it is sent undiminished, though diluted, to the lungs. In these organs it is evident that in the case of alcohol some escapes with the breath, and probable that some may be transformed by oxidization. As much as emerges by the pulmonary veins is poured into the left side of the heart, in contact with which, and with the walls of the systemic arteries, it is brought before its final distribution. It is now uniformly mixed with the systemic arterial blood, and is with it impartially distributed to every part of the body. If subsequently to this part of its course it affect one organ more than another, it is from the susceptibility of the structure not from the direction of the poison.

Stomach.

The changes in the mucous membrane of the stomach are not patent enough to common observation to take the place in this enumeration to which their frequency would undoubtedly entitle them. It was noticed that in three of the

alcoholic series simple ulcer of the stomach existed; in two of the non-alcoholic malignant disease.

Liver.

With regard to this organ the annexed abstract will show that there is in the alcoholic class a striking excess of disease, especially of the fatty and fibroid kinds.

Obvious fattiness of the liver, with or without enlargement or congestion, existed in a ratio of exactly three of the alcoholic to two of the non-alcoholic class.

Abstract showing condition of liver in 149 *alcoholic traders as compared with the same number of non-alcoholic traders.*

		Alcoholic.	Non-alcoholic.
Natural	...	64	79
Hyperæmia	Congested	9	18
	Congested and enlarged	3	2
Fatty change	Fatty	12	11
	Fatty and enlarged	11	3
	Fatty and congested	4	4
	Soft	8	0
Lardaceous	...	4	4
Fibroid increase	Capsule thickened	1	3
	Capsule thickened, structure congested	1	1
	Surface puckered, organs enlarged	0	1
	Surface puckered, organs congested	0	1
	*Early or slight cirrhosis	10	5
	Advanced or well-marked cirrhosis	10	3
	Cirrhosed, fatty, and enlarged	1	0
	Cirrhosed, also containing hydatids	1	0
	Lobules conspicuous	1	0
	Diminished in size (simply)	1	1
	Simply enlarged	7	5
New formations, &c.	Abscesses, pyæmic or dysenteric	1	5
	Tubercle	3	1
	Cancer	1	2
	Hydatids	1	0

* This includes cases where the surface was roughened, and the section showed increase of fibrous tissue or globulation of structure.

Analysis of hepatic symptoms referred to in preceding cases.

		Alcoholic.		Non-alcoholic.
Decided hepatic symptoms were observed in	...	13	...	3
Jaundice (hepatic) ,,	...	9	...	3
Ascites (hepatic) ,,	...	5	...	1
Hæmatemesis (hepatic) ,,	...	1	...	0

Fibroid increase, which in an advanced stage produces the characteristic appearance of cirrhosis, preponderates even more heavily. In the alcoholic series were 24 instances in which thickening of the capsule, puckering of the surface, or early or advanced cirrhosis gave evidence of the change. In the contrasted series the total was 14.

Early cirrhosis among the alcoholic existed in 10 cases, advanced cirrhosis in 12. Among the non-alcoholic early cirrhosis existed in 5, advanced cirrhosis but in 3. Thus, on one side we have 22 instances of cirrhosis, against 8 on the other; in other words the liability to cirrhosis is nearly trebled by a liquorous pursuit.

The production of cirrhosis by alcohol, like most beliefs in medicine whether true or false, has been of late disputed. If there be any who still halt between two opinions in this matter the facts stated can scarcely fail to bring them back to the old view.

With regard to the relative effects of beer and spirits in causing disease of the liver it appears that of the 22 cases of cirrhosis referred to 15 concerned waiters or potmen; 3 cellarmen, 1 a brewer, who was notorious as a spirit drinker, while the remaining 3 occurred in brewers and draymen. Taking brewers and draymen as the representatives of uncomplicated beer drinking it seems that considering the smallness of their number they present nearly their fair share of cirrhosis as compared with the other alcoholic traders. It may be observed, however, that cellarmen who probably may be classed as drinkers of wine and spirit display a much larger proportion of cirrhosis than the beer drinkers.

With regard to beer drinkers it was noted that in two of

the cases the liver was fatty as well as cirrhosed, in one instance the organ weighed no less than 7 lbs. 3 ozs.

As to simple fatty change with enlargement, which, also, is a clear result of an alcoholic occupation, of the 11 persons presenting this alteration only one—a brewer—had been especially concerned with beer. The rest were potmen, waiters, cellarmen, barmen, and barmaids.

Thus the facts, though they do not enable us to distinguish one liquor from another in its morbid action, assert beyond question that alcoholic drink causes both cirrhosis and fatty deposition, or, in other words, increases both the fibroid and the fatty elements of the liver.

Lungs.

Next to the liver in the course of imbibed alcohol come the lungs. Here the more important inferences relate to pneumonia, pleurisy and tubercle.

Pneumonia or hepatization of the lungs, with or without pleurisy, was found in 8 persons of the alcoholic, in 12 of the non-alcoholic series. Pneumonia, as Dr. Stokes pointed out, is no uncommon associate of delirium tremens. The hepatization is usually grey or suppurative and the disease not unfrequently masked. This undoubted concurrence would lead us to expect a preponderance of pneumonia under alcohol which does not exist. It will be found, however, that in other organs as well as in the lung there is with the alcoholic trade a lessened tendency to acute inflammation.

With regard to pleurisy the chief difference to be observed is the prevalence of empyema among the traders in liquor; this as will be afterwards seen accords with a general suppurative tendency which belongs to the class, made manifest by the products of serous inflammations and the results of injuries.

Abstract showing condition of lungs in 149 *alcoholic and* 149 *non-alcoholic traders.*

		Alcoholic.		Non-alcoholic.
	Natural...	20	...	34
Congestion, &c.	Congested posteriorly or otherwise	16	...	16
	Congestion, &c., from typhoid ...	2	...	2
	Œdema (frothy or serous infiltration)	7	...	5
	Emphysema	3	...	3
	Congestion + œdema ...	6	...	5
	Congestion + emphysema ...	4	...	2
	Œdema + emphysema ...	1	...	2
	Congestion + œdema + emphysema	2	...	0
Bronchitis, &c.	Bronchitis (uncomplicated by other pulmonary change) ...	1	...	0
	Bronchitis + œdema ...	0	...	2
	Bronchitis + emphysema ...	0	...	2
Pneumonia and Pleurisy	Pneumonia	1	...	3
	Pneumonia + casts in bronchi...	0	...	1
	Pneumonia + few tubercles, cheesy or cretaceous masses, no tubercles found in other organs ...	3	...	3
	Pleuro-pneumonia	3	...	6
	Pleuro-pneumonia from injury ...	1	...	0
	Pleurisy, recent, acute ...	4	...	5
	Pleurisy + few tubercles (no tubercles elsewhere) ...	2	...	0
	Pleurisy + few tubercles (tubercular disease in other organs)...	0	...	2
	Empyema	3	...	0
	Hydrothorax	4	...	5
Tubercle, &c.*	Miliary tubercles, no vomicæ (no tubercle elsewhere) ...	4	...	3
	Miliary tubercles, no vomicæ (tubercle elsewhere)... ...	3	...	3
	Crude or cheesy tubercle, no vomicæ (no tubercle elsewhere) ...	4	...	2
	Crude or cheesy tubercle, no vomicæ (tubercle elsewhere) ...	2	...	1
	Tubercles and vomicæ (no tubercles elsewhere)	11†	...	12
	Tubercles and vomicæ (tubercular disease elsewhere) ...	23	...	13
	Cicatrix at apex (no tubercle elsewhere)	1	...	0
	Cretaceous mass (no tubercle elsewhere)	6	...	4
	Cretaceous mass (tubercular disease elsewhere)	2	...	0

* With regard to cirrhosis of the lung this comparatively rare condition is not described in any instance. At the time the earlier notes were written the condition was scarcely recognised.

Two instances only occurred in the tables in which tubercle was found in

	Alcoholic.		Non-alcoholic
Pulmonary apoplexy ...	2	...	5
Pulmonary apoplexy + clot in pulmonary artery ...	1	...	0
Pulmonary apoplexy + cretaceous mass + pleurisy (no tubercle elsewhere)	0	...	1
Pyæmic abscess or pneumonia ...	7	...	6
Abscess from unexplained cause	0	...	1

The most important question with regard to the lungs relates to the distribution of tubercle, a point to which further interest attaches in consequence of the differences of opinion which have prevailed as to the relation between alcohol and phthisis. Dr. Peter of New York, whose conclusions though since attacked have been accepted by Dr. Walshe, inferred that drinking protects from tubercle.

Dr. Walshe,* indeed, whose opinion will be seen to be directly opposed to the observations here recorded, says "that publicans, who unquestionably as a class largely consume their own vendibles, are *cæteris paribus* less destroyed by phthisis than persons in various other walks of life." The antagonism of tubercle and alcohol appears to have been widely believed though apparently on insufficient evidence. Trousseau,† however, states that his experience has led him to coincide with those who hold the contrary view, and the facts before us instead of supporting strongly contradict the view that alcohol is antagonistic to tubercle. In tuberculosis we see, if the facts adduced be trustworthy, one of the modes in which the body is most vulnerable to drink.

Mindful of differences of opinion as to the nature and varieties of tubercle, I have as far as practicable specified with

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other organs, none being in the lungs. Both were in the non-alcoholic series; one was of miliary subpleural tubercle, the other tubercle of brain.

† One of these was associated with diabetes. Pneumonia, bronchitis, and pleurisy are not mentioned when in connection with extensive tubercular disease of the lung. The same statement applies to emphysema, fibroid and other change clearly secondary to the tubercular disease.

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* 'Diseases of the Lungs,' 4th edition, p. 453.

† The results of this paper nearly coincide, in most respects, with Trousseau's views.

regard to each case not only the nature of the pulmonary change but also the presence or absence of any conditions recognised as tubercular in any other part of the body. Doubts which may concern a change limited to the lung, as between tubercle, degenerating fibrosis or caseating pneumonia, can find no place where there are characteristic deposits or ulcers elsewhere. Community of tubercle-like changes to several organs may be held as sufficient indication of their truly tubercular character, at the same time that their being limited to one does not necessarily clear them from the imputation.

Taking first the more doubtful class, changes apparently tubercular but limited to the lung, such, comprising miliary, crude and cheesy tubercles, vomicæ, cicatrices and cretaceous masses, were found in an aggregate of 31 persons of the alcoholic, in 25 of the non-alcoholic.

The same changes in the lungs, certified as tubercular by the existence of tubercular disease in other organs, were present in 30 individuals of the alcoholic, in 19 of the non-alcoholic series.

Thus under alcohol reputed tubercle of the lung of every kind preponderates, and the preponderance is greatest where the tubercular nature of the change is the least doubtful. It will be observed that the typical form of tubercular phthisis with tubercle and vomicæ in the lungs and tubercular disease in other organs occurred in 23 of the alcoholic to 13 of the contrasted series.

Including all kinds, reputed tubercle of the lung affected 61 persons of the alcoholic, 44 of the non-alcoholic class; nearly 3 to 2. This must be conclusive as to the fact that drink instead of preventing promotes phthisis.

It is believed, probably with truth, that alcohol causes pulmonary fibrosis. The cases before us, however, gave no instance to which the terms cirrhosis of the lung or fibroid phthisis could be confidently applied. Within the necessary limits of this inquiry the amount of fibroid associated with the tubercle-like changes in each instance could not be indicated. Care has been taken, however, to separate those

cases in which other organs participated in the tubercular change from those in which the lung suffered alone. Any case of simple fibroid or cirrhotic disease which may have occured has necessarily gone to swell the number of the latter class.

Tubercle.

After what has been said with regard to pulmonary tuberculosis, the wider question of tubercle without reference to its seat shrinks into a mere corollary. Only two instances were recorded, one of sub-pleural, one of cerebral tubercle, in which the lungs did not share in the tubercular disease. Both were in the non-alcoholic series, placing the sum of tubercular persons at 61 alcoholic; 46 non-alcoholic.

The question arises whether the prevalence of tubercular disease in the lung is to be interpreted as especially pulmonary, or especially tubercular? Is the tendency to tuberculosis regardless of its seat, or to disease local to the lung out of which the tubercle is engendered? It is clear that the tendency to tubercle is general since it preponderates in other organs even more than in the lung. In each part of the body amenable to tubercle, brain, liver, kidneys, bowels, and peritoneum, the frequency of its occurrence is at least doubled by the alcoholic pursuit. Attributing the excess, as we need must to the influence of the liquor, we arrive at an important and secure deduction that alcohol engenders tubercle.

Tubercular disease in organs other than the lungs.

	Alcoholic.		Non-alcoholic.
Brain	6	...	3
Liver	3	...	1
Kidneys	8	...	4
Spleen	1	...	0
Bowels (tubercular ulceration) .	12	...	5
Mesenteric glands . .	5	...	2
Peritoneum . . .	2	...	0
	37		15

Heart.

In the alcoholic series atheroma and fatty degeneration prevail; in the non-alcoholic, endocarditis and pericarditis. Speaking roughly alcoholic pursuits increase the degenerative, and diminish the inflammatory tendency.

Taking atheroma first as often the initial change, it will be seen that labourers in liquor, notwithstanding that they die younger, are the more prone to this deterioration. Atheroma of the aorta in particular was noted in 39 persons of the alcoholic, in 29 of the non-alcoholic series, a proportion of about 4 to 3. Aneurism and sanguineous apoplexy connected as they are with atheroma, preponderate on the same side.

Abstract showing condition of heart in 149 *alcoholic and* 149 *non-alcoholic traders.*

		Alcoholic.	Non-alcoholic.
	Healthy	57	67
Pericardium, &c.*	White patch on surface ...	8	7
	White patch + valvular disease .	1	0
	White patch + valvular disease + hypertrophy	2	1
	Pericardial adhesions ...	2	2
	Pericardial adhesions + valvular disease	2	3
	Pericardial adhesions + valvular disease + hypertrophy ...	1	3
	Recent pericarditis (lymph and serum)	2	2
	Recent pericarditis (pus and sero-purulent fluid)	3	0
	Recent + old pericarditis ...	0	2
	Recent pericarditis + hypertrophy	3	1
	Recent pericarditis + endocarditis	1	1
	Recent pericarditis + old valvular disease	0	2
Valves, &c.	Valvular disease	11	14
	Valvular disease + hypertrophy .	5	9
	Valvular disease + hypertrophy + dilatation	0	3
	Valvular disease + hypertrophy + fatty change	1	2
	Valvular disease + fatty change	1	1
	Valvular disease + dilatation ...	1	1

* Fluid in pericardium is not recorded; when the result of inflammation it appears under the heading of pericarditis.

		Alcoholic.		Non-Alcoholic.
Muscular wall, &c.	Simple hypertrophy (*i.e.* unconnected with other cardiac or obvious arterial change) ...	12	...	5
	Hypertrophy + white patch on surface	1	...	1
	Hypertrophy + recent endocarditis	1	...	0
	Hypertrophy + aortic disease ...	4	...	1
	Dilatation	7	...	5
	Attenuation, flabbiness, or flaccidity	10	...	9
	Fatty	6	...	4
	Fatty + enlarged	2	...	0
	Fat on surface	2	...	1
Aneurism, &c.	Aneurism of aorta	2	...	0
	Aneurism of aorta + valvular disease	1	...	0
	Aneurism of coronary arteries + valvular disease (embolism) ...	0	...	1
	Aneurism of ventricle + pericarditis + valvular disease + hypertrophy	0	...	1

Atheroma in alcoholic and non-alcoholic traders (149 *of each*).

Atheroma of valves	3	...	7
„ coronary arteries	1	...	2
„ aorta	25	...	20
„ cerebral arteries	3	...	1
„ valves + coronary arteries ...	0	...	1
„ valves + aorta	10	...	6
„ coronary arteries + aorta ...	3	...	1
„ coronary arteries + cerebral arteries	1	...	0
„ aorta + cerebral arteries ...	1	...	1
„ aorta + valves + cerebral arteries	0	...	1
Total number of individuals in which atheroma was noted	47	...	40

With regard to valvular disease the stress is in the contrary direction. Valvular disease, speaking inclusively without distinction of kind, affected 28 persons concerned with drink, 42 concerned otherwise, being less frequent with drink as 2 to 3. It was not practicable in the tabulations to distinguish the results of valvular atheroma from those of endocar-

ditis; if, however, atheroma on the valves, as elsewhere, is more frequent under liquor, the preponderance of endocarditis in the non-alcoholic series is proportionally increased. In any case it is clear that inflammatory affections of the valves are greatly the less frequent in the drink-trades.

Pericarditis, evinced by recent lymph, purulent fluid, or adhesions, occurred in 14 persons of the alcoholic, 16 of the non-alcoholic class. Thus, in one shape or another the disorder falls pretty evenly, though we find differences when we take into account the diverse circumstances in which the serous inflammation arises. Pericarditis associated with valvular disease, often though not necessarily rheumatic, left its traces in lymph or adhesions in 4 of the alcoholic, 9 of the opposed series, the preponderance corresponding with that of valvular disease. Recent pericarditis, with simple hypertrophy, always renal, was more common with the alcoholic, the numbers being 3 to 1. The suppurative form of the disorder was only found in the alcoholic class; of this there were three instances. This has importance in connection with the prevalence of suppurative pleurisy under the same influence of an alcoholic pursuit.

White patches upon the heart which probably are sometimes though not always due to pericarditis, are slightly more frequent in the alcoholic. This is in accordance with an opinion which has been espoused by Lancereaux to the effect that these marks are increased in frequency by the use of alcohol.

With regard to changes in the muscular tissue of the heart, fatty degeneration or superficial excess of fat occurred in 12 of the alcoholic, in 8 of the non-alcoholic series, the fatty change prevailing in the heart as in the liver in the the class where drink was at hand.

Simple hypertrophy or hypertrophy without tangible cause in the vascular system was more frequent with liquor, as 15 to 6; more than twice as common in the alcoholic as in the other class. Such hypertrophy is generally regarded as renal, and it is of interest to inquire whether its marked preponderance is to be traced to a corresponding increase of renal disease under the influence of drink. I have, therefore,

shown in detail the condition of the kidneys in every case of simple cardiac hypertrophy.

Condition of kidneys accompanying hypertrophy of the heart, either simple or associated only with recent pericarditis.

Alcoholic series—12 cases simple hypertrophy + 3 cases hypertrophy with pericarditis = 15 cases.

Non-alcoholic series—5 cases simple hypertrophy + 1 case hypertrophy with pericarditis = 6 cases.

Kidneys.

		Alcoholic.		Non-alcoholic.
Intertubal.	Advanced granular degeneration	5	...	3
	Early or slight granular degeneration	2	...	0
	Cysted, otherwise natural ...	1	...	0
Slight or uncertain.	Coarse, large and coarse, or coarse and fatty	3	...	0
Tubal.	Natural size, mottled, grey deposit in cones	0	...	1
	Large, solid, mottled ...	0	...	1
	Large and congested ...	2	...	0
	Healthy	2	...	1
		15	...	6

It would seem that simple hypertrophy of the heart may be associated not only with obviously granular kidneys but with kidneys otherwise and variously altered, and also with kidneys which passed as natural. The preponderance of hypertrophy of the left ventricle under alcohol was not solely due to a preponderance of renal disease, notwithstanding that granular degeneration with consequent hypertrophy is more common with liquor than without. In more than half the cases of this form of cardiac hypertrophy the kidneys were either natural or were described as congested, fatty or coarse, conditions which could not be confidently regarded as enough to produce cardiac change. We may probably conclude that what is roughly termed simple hypertrophy,

though often dependent on renal disease, has its origin also in deteriorations of the minute arteries, such as have been recently described before this Society,* which are not necessarily accompanied by obvious renal deterioration.

These minute arterial changes, if we may accept hypertrophy of the left ventricle as their index, prevail among the alcoholic to a larger extent than does renal disease, and must, therefore, be attributed to the direct influence of the liquor upon the blood-vessels.

Nervous system.

It is unnecessary to repeat that by the rule of classification delirium tremens is confined to one series, this disease having been taken as sufficient evidence of excess in drink to exclude its subjects from the non-alcoholic category.

Of the alcoholic traders, 17 or about 1 in 9 were affected with this disease, proof enough if it were wanted of the habits of the class.

In other respects the alcoholic side displayed an excess of inflammatory and hæmorrhagic affections of the brain, and of cerebral uræmia. The preponderance was most striking with regard to inflammatory conditions, and was marked whether associated with or independent of tubercle.

Six cases of tubercular disease of the brain occurred in the alcoholic, 3 in the non-alcoholic series.

Acute inflammation of the brain, not connected with tubercle, described as cerebro-spinal meningitis, encephalitis, and inflammatory softening, occurred in four instances, all of which belonged to the alcoholic series. There is evidence to show that drunkenness is alone sufficient to set up meningitis in an acute form. One of the cases in the table was a striking example of this, and another came under my own observation. In neither was there any tubercular or other disease with which the attack could be associated.

* Paper by Sir W. Gull and Dr. Sutton in the preceding volume.

There is also in the same category a strong disposition to the collection of aqueous fluid in the intercranial spaces; this, not usually manifest during life, is probably often simply a compensation for chronic shrinking of the cerebral matter.

An alcoholic trade increases the liability to cerebral hæmorrhage. Putting aside a case of cerebral purpura the tables include 10 cases of cerebral hæmorrhage of the ordinary kind. In every case the kidneys were granular, and, no doubt, with the same regularity the vessels of the brain were diseased. Six of these cases occurred in the alcoholic, 4 in the non-alcoholic series. Thus, cerebral extravasation, like hypertrophy of the heart, both especially associated with renal disease and both taking their origin in vascular decay, are both fostered by alcohol.

Disturbances of the brain dependent on uræmia are also more frequent under alcohol; but this fact will be more appropriately considered in connection with renal disease.

Abstract showing state of brain and nervous system in 149 *alcoholic and* 149 *non-alcoholic traders.*

		Alcoholic.		Non-alcoholic.
Hæmorrhage.	Sanguineous apoplexy ..	6	...	3
	Purpuric hæmorrhage under arachnoid	1	...	0
	Slight hæmorrhage under arachnoid, with congestion of brain, with granular kidney ...	0	...	1
Delirium tremens.	Delirium tremens	15	...	0
	Delirium tremens + calcareous deposit (in falx)	1	...	0
	Delirium tremens + uræmia (epileptiform convulsions) ...	1	...	0
Uræmia.	Uræmia (marked during life by convulsive attack, or partial or complete coma)	5	...	3
	Excess of fluid with anæmia, associated with renal disease, but without marked uræmic symptoms	2	...	3

		Alcoholic.	Non-alcoholic.
Excess of watery fluid, &c.	Excess of fluid in and about brain, without cerebral symptoms after death by cholera, fever, carbuncle, erysipelas, and suicide	5 ...	1
	Excess of fluid after death from acute rheumatism + bony plate in arachnoid	0 ...	1
	Congestion, or blood-tinged fluid under arachnoid, with erysipelas	1 ...	1
	Excess of fluid in and about brain, productive of symptoms and due to disease primarily of brain	2 ...	3
Tubercle.	Tubercular meningitis ...	4 ...	2
	Tubercular disease of brain ...	2 ...	1
Inflammation.	Inflammation of brain or membranes from blow or injury ...	3 ...	3
	Inflammation of brain or membranes from disease of skull ...	1 ...	1
	Cerebro-spinal meningitis ...	1 ...	0
	Acute encephalitis (from hard drinking); softening, with, in one case, pus in ventricles, in the other lymph under arachnoid	2 ...	0
	Softening + excess of fluid + congestion (cerebral symptoms)	1 ...	0
	Softening of brain, circumscribed (pyæmic?)	0 ...	1
	Softening of brain with symptoms of cerebral disease ...	1 ...	0
	Old adhesions in arachnoid + insanity	1 ...	0
	Coagula (ante mortem) in arteries	0 ...	1
	Calcareous or osseous matter in membrane or choroid ...	0 ...	1
	Tumour of brain	1 ...	1
	Enlarged Pacchionian bodies ...	1 ...	0
	Epilepsy	1 ...	0
	Epilepsy + tetanus ...	1 ...	0
	Paraplegia from disease of spine	1 ...	1
	Paraplegia from disease primarily of cord	0 ...	2

Condition of kidneys associated with extravasation of blood within the cranium.

Alcoholic series.

Common sanguineous apoplexy, 6 cases :

Kidneys, advanced granular in	...	... 4
„ slightly „	...	... 2
		6

Purpuric hæmorrhage, 1 case :
Kidneys natural.

Non-alcoholic series.

Common sanguineous apoplexy, 4 cases :

Kidneys advanced granular in	...	... 3
„ slightly „	...	... 1
		4

Results of accidents and injuries.

It is well known that with drunkards the power of healing is weakened, that processes of repair are slowly and unsafely conducted, and that small injuries and trivial operations are attended with disproportionate danger. The facts before us bear upon this point.

Traders in liquor are less exposed in their way of life to external hurts than those to which they are compared. Draymen meet with horse accidents, and potmen are sometimes involved in disputations which end in a violent misuse of the pewter pot, but the class to which they belong is less often subjected to accidental injury though, possibly, suffering more fatally from its effects, than the other. This is shown by the distribution of necessarily and immediately fatal accidents. Such accidents as are inevitably fatal, and are therefore unaffected in their issue by the antecedents of the victims, will by the number on either side indicate the liability of the class to violent bodily damage. We find five necessarily fatal accidents in the alcoholic, ten in the non-alcoholic series. Thus, it would seem that traders in drink are only half as often the subjects of such accidents as those

who follow other and in this respect more dangerous callings. If the same rule applies to small accidents as to great, traders in liquor have a comparatively small exposure to mechanical violence. But accidents among them, though less frequent, are more fatal.

List of fatal accidents.

Alcoholic.	Non-alcoholic.
5 — *Directly fatal.*	10
Fracture of ribs, laceration of lung.	Fracture of skull.
Fracture of pelvis.	Fracture of thigh, &c.
Cut throat (suicidal).	Rupture of bowel.
Fracture of skull.	Fracture of skull.
Laceration of liver.	Laceration of liver.
	Gunshot (suicidal).
	Fracture of skull.
	Laceration of liver.
	Fracture of leg, &c.
	Wound of brachial artery.
10 — *Succeeded by delirium tremens.*	0
Bruise of chest.	
Burn, ulcer of duodenum.	
Compound fracture of leg.	
Slight injury of nose.	
Cut wrist.	
Dislocations of ankle.	
Compound fracture of leg.	
Slight bruise of head.	
4 — *Succeeded by pyæmia.*	4
Compound fracture of leg; amputation.	Compound fracture of leg.
Scalp wound, pus outside dura mater, &c.	Abscess of thumb, followed by cellulitis.
Compound fracture of leg.	Crushed hand, amputation.
Compound fracture of thigh.	Compound fracture of skull.
5 — *Succeeded by diffuse cellulitis.*	3
Scalp wound.	Scalp wound, fracture of spine.
Slight injury of thigh (delirium tremens occurred also).	Fracture of skull, laceration of brain.
Fracture of thigh, ununited.	Severely cut throat.
Bite of donkey.	
Pugilistic bruises.	
1 — *Succeeded by erysipelas.*	1
Injury of leg, suppuration of knee, &c.	Small scalp wound.
1 — *Succeeded by gangrene.*	0
Fracture of fingers.	

Alcoholic.		Non-alcoholic.
2	*Succeeded by empyema and pleuro-pneumonia.*	0
Simple fracture of ribs. Fracture of ribs, &c.		
1	*Succeeded by tetanus.*	0
Burn.		
1	*Succeeded by superficial softening of brain.*	0
Blow on head.		
1	*Succeeded by cystitis.*	0
Fracture of leg.		

Pyæmia seizes more than its share.

Diffuse inflammation of the cellular tissue is fatal and readily induced.

Complications of accidents ultimately, but not immediately or necessarily fatal.

	Alcoholic.	Non-alcoholic.
Pyæmia	4 ...	4
Cellulitis	5 ...	3
Erysipelas	1 ...	1
Gangrene	1 ...	0
Pleuro-pneumonia } from broken ribs ...	1 ...	0
Empyema } from broken ribs ...	1 ...	0
Pus between skull and dura mater (from scalp wound)	1 ...	0
Tetanus	1 ...	0
Superficial softening of brain (from blow on head)	1 ...	0
Accidents directly and necessarily fatal ...	5 ...	10

The non-alcoholic series yielded three fatal cases of cellulitis which were respectively produced by fractures of the skull and spine and a deeply cut throat. The alcoholic series gave five, one of which only had its origin in a severe injury, namely, a fracture of the thigh which had refused to unite; the others were consequent upon a scalp wound, a slight injury of the thigh, pugilistic bruises, and the bite of a donkey.

An increased liability to spreading inflammation of the cellular tissue belongs, therefore, to the trader in drink, produced, no doubt, by the liquor. In other words, under the

chronic influence of alcohol, the product of inflammation is apt to be purulent and non-adhesive, such as can spread and disseminate, rather than of the plastic and circumscribing sort, which limits the process by means of fibrillating lymph, and eventuates in false membrane and solid repair. The formation of pus instead of plastic lymph under the same influence is seen in the generation of empyema and of abscess within the skull from external injuries, nothing of the kind having occurred in the non-alcoholic category. These observations have additional weight when taken with reference to what has already been shown with regard to the distribution of the suppurative forms of pleurisy and pericarditis.

These conclusions are consistent with some observations lately published by Dr. Péronne,* who reckons among the effects of chronic alcoholism in relation to the healing of wound, abundant suppuration, diffuse inflammation, partial gangrene, secondary hæmorrhage, and tardiness of healing.

Kidneys.

The effect of alcohol upon the common channels of exit may naturally be considered last. The kidney, which has been credited with a morbid susceptibility to alcohol much in excess of what is warranted by facts, is the only gland of excretion which need occupy us in this relation.

By reference to the annexed abstract it will be seen that the kidneys were described as free from disease in almost exactly the same number of the two classes. The same statement holds good with regard to kidneys which presented no further departure from their natural state than simple congestion, probably in many instances a fleeting and almost accidental condition. Thus it remains that the number of kidneys presenting tangible morbid changes was almost exactly the same on the two sides; or, to speak precisely, the kidneys of 82 persons in the alcoholic, of 83 in the non-

* 'De l'Alcoolisme dans ses Rapports avec le Traumatisme,' p. 154.

alcoholic series, presented distinct morbid changes. This statement includes, of course, not only the alterations recognised by albuminuria, but comprehends also suppurative, tubercular, cancerous, embolic, and calculous affections.

Condition of kidneys in 149 *cases of each kind.*

		Alcoholic.	Non-alcoholic
	Natural...	49	48
Hyperæmia.	Congested	18	18
	Congested and enlarged	10	5
Tubal changes.	Coarse ...	3	3
	Coarse and enlarged	4	1
	Slight or uncertain change in cortex	2	4
	Large, smooth, mottled*	5	9
	Mottled, normal size, grey deposit in cones	0	1
	Smooth, pale, yellow or grey, cortex shrunk	1	1
Fatty change.	Fatty or flabby	2	1
	Fatty or flabby and enlarged	3	3
	Lardaceous	3	6
Intertubal, fibroid increase.	Cysts without other change	1	2
	Cysts and depressions	3	1
	Cysts and tubercles...	0	1
	Slightly granular	10	11
	Highly granular	18	15
	Granular + pyelitis	1	0
	Granular + tubercles	1	0
	Granular + conversion of other kidney into a cretaceous mass...	0	1
	Granular + stone ...	1	0
	Pyelitis...	0	2
	Abscesses in kidney associated with pyelitis	1	6
	Abscesses from pyæmia	1	1
	Abscess of uncertain nature	1	0
	Tubercular disease ...	8	4
	One converted into cretaceous mass, other natural	0	1
	Cancer ...	1	1
	Fibrinous blocks, without other change	0	2
	Stone ...	2	1

* It is possible that some of the kidneys thus described in the earlier records were lardaceous, in which case the preponderance of lardaceous disease in the non-alcoholic series would probably be greater than is represented.

*Symptoms referable to albuminuria in the preceding cases.**

	Alcoholic.	Non-alcoholic.
Number of cases in which renal symptoms of any kind were prominent	20	26
Renal œdema occurred in	14	18
Internal dropsy, apparently renal, occurred in	4	6
Albuminuria found in	23	29
Cerebral uræmia (coma, convulsions, &c.) ...	5	3
Death from renal disease (directly)† ...	10	14

Descending to details, and taking first changes which, so far as the descriptions allow us to judge, are in the tubes and their contents, we find on adding the kidneys described as enlarged and congested, and those which were fatty, to the varieties included within the tubal bracket, that in one or other of these ways the kidneys were diseased in 30 persons of the alcoholic series to 28 of the opposed class. Among these changes those only which had marked predominance in the alcoholic category were enlargement with either coarseness or congestion; of these were 14 in the alcoholic, to 6 in the contrasted series. The typical large white kidney was most common in the non-alcoholic class. Thus, it would seem that though certain alterations, which apparently have their seat within the tubes, prevail under an alcoholic trade, yet that they are not such as have marked symptoms or are generally discoverable during life. Under this influence tubal changes, though slightly more frequent, are less acute and obvious. It must be taken into account, however, as affording a partial explanation of their lessened liability to acute inflammatory affections of the kidney, that traders in liquor are less exposed to wet and cold, not infre-

* The table comprises all the renal symptoms observed in the cases of the two series, excepting those referred to pyelitis or renal abscess, tubercle, or other morbid growths, or calculus. These are excluded for the sake of placing the results of albuminuria in a distinct form.

† Sanguineous apoplexy is not included here.

quent causes of acute nephritis, than are the persons with whom they are compared. (See page 31.)

With regard to the intertubal or fibroid change we find distinct granulation in 31 alcoholic to 27 of the non-alcoholic series. If we look upon cysts (as with general truth we may) as evidence also of intertubal change, we must place the total among the alcoholic at 35, among the non-alcoholic at 31. Thus, it would seem that the frequency of granular degeneration is slightly increased by a conversance with liquor, the inequality in the distribution of the disease between the two classes being nearly represented by the proportion of 8 to 7.

Lardaceous infiltration, like other diseases of the kidney, has been loosely ascribed to intemperance in liquor. This change, however, is decidedly the less frequent in the alcoholic series. The diminution may be due, wholly or in part, to the comparative rarity of surgical accidents in the class, with a correspondingly smaller share of such hurts of bone and joint as give rise to long suppuration. We may at least conclude that the excessive imbibition which accompanies habitual intimacy with liquor, though it may not prevent, does nothing towards engendering the change in question.

This evidence, relating solely to inspection of the kidneys, may be supplemented by collateral reference to other organs affected in dependence upon or association with them, as well as by a glance at the accompanying symptoms. Pathological facts as gathered after death and clinical observations made during life agree with each other and with the preceding statements in indicating under alcohol an increase of granular degeneration, a diminution of active tubal nephritis and of lardaceous disease.

As to the changes in the vascular system we find under alcohol a marked excess of common atheroma, of simple hypertrophy of the heart and of sanguineous apoplexy. These indications of vascular deterioration are so often associated with the granular kidney that they may be generally taken as signs of its prevalence. Simple hypertrophy, however, and cerebral extravasation predomina

the alcoholic series to an extent which would lead us to expect a larger excess of renal disease than is found. Alcohol has, it would seem, independently of renal disease, a damaging effect upon the arteries, whence the heart may become hypertrophied though the kidneys are natural. Thus, the deterioration of the coats of the vessels which accompanies renal disease, more especially of the granular kind, is enhanced by familiarity with liquor.

Cerebral uræmia, which is a more constant associate of the granular kidney than of the other forms of renal disease, also predominates in the alcoholic series.

Symptoms which point to other renal changes tell with equal consistency a different tale. In the alcoholic series we note fewer cases in which renal disease was detected during life; there is here less discovered albuminuria, less dropsical effusion of every kind, and a smaller number of deaths attributable directly to renal mischief. These details concur with the facts derived from examination of the kidney itself in showing that traders in liquor, either from the operation of the drink or the circumstances of the trade, are less liable than their neighbours to lardaceous disease and to tubal inflammation of the active and prominent kind.

Diverging for a moment from the strict subjects of our inquiry, and looking at the state of the kidneys generally accompanying delirium tremens, we may find additional evidence on this point.

I subjoin a table* compiled from the same sources in which the post-mortem state of the kidney in subjects of delirium tremens is compared with that of men not known to have been intemperate who have been cut off by accidental violence.

* This table is similar to one published in my work on 'Albuminuria;' it, however, comprises the records of seven additional years.

Kidneys after death with delirium tremens, and from accident without known intemperance. All of adult males. (From the year 1841 *to* 1871 *inclusive.)*

Condition of kidneys.	Delirium tremens. 58 cases.	Accident. 58 cases.
Natural	28 ...	24
Congested	15 ...	7
Slight or uncertain change in cortex	5 ...	1
Large, smooth, mottled ...	3 ...	1
Granular surfaces	6 ...	8
Cysts without other change ...	1 ...	7

Average age of delirium tremens patients, 38 years.
„ accident „ 41 „

Assuming, as we safely may, that most of the victims of delirium tremens have been habituated to alcoholic excess, we find in this fraternity of drunkards what to many persons may be a surprisingly small increase of renal disease. It must be observed, however, that the cases of accidental death are taken without exclusion as to trade, so that a certain proportion of lead workers, of whose calling granular degeneration is a necessary result, have been admitted.

Taking all the facts together, and allowing to the trader in liquor his full measure of protection from weather and violence, we must conclude—

1. That alcohol has no effect in causing lardaceous disease.

2. That it promotes granular degeneration, though not to an extent commensurate with the arterial change produced by the same agent.

3. That acute nephritis, though in exceptional instances possibly caused by intense alcoholic excess, is not generally increased in frequency by habits of drinking. In its comparative rarity under this influence the affection resembles others, save of the brain, of the acute inflammatory type.

4. Chronic and latent tubal disturbances, including fatty degeneration, are increased by the use of alcoholic liquors.

5. And that on the whole the kidneys are affected less injuriously by the popular poison than are the liver, the lungs, the blood-vessels, and the nervous system.

General conclusions.

We are led to conclusions which are mainly with, though in some respects against, old views. In the present state of medicine it may be as useful to fortify old truths as to assault old errors. The results may be thus summed up.

Persons who trade in liquor drink on an average more than those who do not, and their morbid peculiarities are mainly due to that excess. Estimating the effects of alcohol on this basis by means of comparison between the class described and persons similarly situated save in relation to liquor, the following conclusions have been reached.

Alcohol shortens life; to trade in liquor costs three and a half years.

Reviewing the morbid results which the examination of each organ has revealed, they present a consistency which is in some sort their warrant.

Alcohol causes fatty infiltration and fibroid encroachment; it engenders tubercle, encourages suppuration, and retards healing; it produces untimely atheroma, invites hæmorrhage, and anticipates age.

The most constant fatty change, a replacement by oil of the material of epithelial cells and muscular fibres, though probably nearly universal, is most noticeable in the liver, the heart, and the kidney.

The fibroid increase is little evident in the simply fibrous structures such as fasciæ and tendons, but occurs about the vascular channels and superficial investments of the viscera, where it causes organic atrophy, cirrhosis, and granulation. Of this the liver has the largest share; the lungs are often similarly though less simply affected, the change being variously complicated with or simulative of tubercle; the kidneys suffer in like manner but in a more remote degree.

Alcohol also causes vascular deteriorations which are akin both to the fatty and the fibroid. Besides tangible atheroma there are minute deteriorations of the arterial walls, which show themselves by cardiac hypertrophy and cerebral hæmorrhage.

Drink causes tuberculosis, which is evident not only in the lung, but in every organ which is amenable. It must be observed, however, that from the tendency of tubercle to dissemination it is only necessary that it be planted in one organ to be found in many.

Drink promotes the suppurative at the expense of the adhesive process. This is sufficiently seen in the results of pneumonia, of pleurisy, and of pericarditis, in diffuse inflammation after injuries, and in the sloth and insecurity of the healing process.

Descending from general conditions to individual organs, the effect of alcohol upon the nervous system must be looked upon as special and taken by itself, since nervous matter presents to this agent a singular excitability of function, or in other words, a singular susceptibility of structure, for the purpose of acting upon which mankind in all countries and in all ages have sought and used alcoholic drinks.

Passing over those effects of intemperance which like delirium tremens are manifest rather during life than after death, we find that the brain pays a large reckoning in the shape of inflammation, atrophy, and hæmorrhage.

Alcohol multiplies inflammatory states of the brain of every kind, both of the substance and the envelopes, whether tubercular or not.

It occasions a gradual shrinking of the brain, as evinced by the accumulation of fluid in spaces once filled by cerebral substance.

It causes a liability to cerebral hæmorrhage by means of the arterial deterioration common to the greater part of the body.

With regard, to other organs they are damaged by alcohol much as they stand in its line of absorption. Passing over the stomach, the changes in which, however numerous, are, save when it is ulcerated, more prominent during life than after death, we come to the liver. This organ suffers more than any other, chiefly by way of cirrhosis and fatty impregnation. Next the stress falls upon the lung, probably less heavily than upon the liver, certainly more heavily than

upon any other part of the body with the exclusion of the nervous system. The mischief in the lung takes apparently every shape of phthisis. It is probable that the change most often is of a kind to which the term tubercular would strictly apply, while there is evidence, not comprised in the present tables, of a fibroid overgrowth, which apparently may either accompany or simulate tubercle. The arterial deterioration need not be further mentioned than to assign its relative frequency. Judging by its tangible results—atheroma, hæmorrhage, and cardiac hypertrophy—we should assign to this change a large share in the pathology of intemperance, though unless the connection between alcohol and tubercle has been much overrated it would seem to play a less fatal part than disease of the tubercular kind. Lastly, the kidneys, more remotely exposed, have a smaller participation in the common damage of alcoholism. They undergo congestive enlargement, fatty and fibroid change; they do not suffer, however, commensurately with the blood-vessels or with the same frequency as other viscera.

So far we have seen only the ill which alcohol produces; it may be asked is there none which it obviates? Apart from any medicinal or curative action, which the evidence before us does not touch, has it no *per contra* of prevention? It is difficult to answer this inquiry. Some active inflammations, such as pneumonia and endocarditis, are diminished in the alcoholic trades, but it must at once be seen that the increase of the alcoholic disorders must necessarily produce an apparent diminution of all which are unaffected by this agent. A man may be saved from pneumonia or acute rheumatism, not because alcohol antagonises such diseases, but because it kills him prematurely. He can die but once. Therefore, though under alcohol some forms of disease are comparatively infrequent, we must use much caution in concluding that it has a directly preventive influence. Nevertheless, it may be laid down as an axiom that any drug which can do harm can do good. Disease is most various, and may or rather *must* represent contrary conditions. It may be positive or negative, plus or minus. Too much or too little of any of

the shapes of heat, food, and work may spoil the equipoise of health. If a drug promotes one change it may prevent its opposite. Alcohol certainly gives an asthenic type to disease; although we cannot as yet say with certainty that it defibrinates the blood, yet it retards adhesive and plastic processes. This influence may be beneficent if it hinder the development of acute inflammation, and obviate the formation of coagula where, as in acute rheumatism, that action is harmful. It is possible that by some such antagonism to the sthenic and fibrous type of disease we may explain the remarkable paucity of endocarditis in the alcoholic series. But, at the best, the protecting is less certain and less effective than the deteriorating influence.

In brief and final enumeration, alcohol replaces more actively vital materials by oil and fibrous tissue; it substitutes suppuration for new growth; it promotes caseous and earthy change; it helps time to produce the effects of age; and, in a word, is the genius of degeneration.

ON THE

RESPIRATORY MOVEMENTS IN MAN,

WITH AN

ACCOUNT OF A NEW INSTRUMENT FOR MEASURING THE MOVEMENTS OF THE CHEST.

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THE various movements of breathing have often attracted the attention of observers of the different actions of the human body. Non-medical writers have spoken of their significance as signs of the various emotions. Physiologists and physicians have found in them evidence either of healthy function or of disease.

Diversities in the movements of respiration are often too obvious to require an "experienced eye" or a "practised hand" to detect them. We need only to watch the quick panting breathing of children at play, or their breasts heaving with convulsive sobs, to notice the deep inspirations of professional singers, or the fully expanded, almost motionless, chest of an expectant athlete; and, on the other hand,

a student of disease would readily compare the quick short breath of a consumptive patient with the laboured action of the rigid chest of an asthmatic.

All these variations in the working of the mechanism of the chest are sufficiently plain without the aid of instruments of measurement, but something more is required when they become the subject of careful study. Dr. Whewell has well said "the reference of objects to exact ideas of space, number, position, motion, and the like, is the first step in science." Before we can hope, therefore, to attain to a scientific knowledge of these movements, and of the mode in which they are produced, exact numerical records of their extent under different circumstances are required.

Many attempts have been made to obtain these data, and very various instruments have been devised for the purpose.

Woillez and Piorry simply measured the circumference of the chest with an unyielding band, marking with ink the points corresponding to the middle of the sternum and the spines of the vertebræ.

Chomel and Walsh, Hirtz and Wintrich, used graduated tapes. Montault combined this with a plessimeter and stethoscope, and Dr. Stokes employed callipers with which to measure the different diameters of the chest.

Woillez[1] also invented an ingenious instrument, which he named the "Cyrtomètre," consisting of a jointed band of whalebone, which so retained the shape of the chest that its curves could be transferred to paper, giving correct diagrammatic representations of the contour of the two sides. Woillez was sanguine enough to think that by means of this instrument he could show variations in the perimeter, or in the antero-posterior diameter, at different epochs of such maladies as pulmonary congestion, coming on in the course of acute febrile disorders, in all acute thoracic affections, in eruptive fevers up to the appearance of the rash, and in advancing pleuritic effusions.

[1] 'Rech. pratiques sur l'Inspection et la Mensuration de la Poitrine.' Paris 1840.

Various stethometers have also been devised by Drs. Wintrich, Quain, and Leared. Dr. Sibson has employed his well-known chest measurer, and Dr. Burdon Sanderson his ingenious stetho-cardiograph. Much valuable information has undoubtedly been the result of all these investigations, and many important facts relating to the mechanism of respiration have been settled by them, especially by the elaborate researches of Dr. Sibson in England, M. Bert in France, and MM. Traube and Rosenthal in Germany.

But there is one important defect in all these measurements. Owing to the obliquity and partially circular shape of the ribs, and to their rotation on two axes, it may readily be seen that the motions of any point on the walls of the chest, on either side of the sternum, may, in forced breathing, take place in three planes at right angles to one another. Owing also to the variations even in healthy breathing, it is further important to measure the movements in different directions during one act of breathing. Now, all the modes of measurement so far enumerated simply give either the gross enlargement of the circumference of the chest, or the resultant of the movements of its different parts, without estimating their extent in the several directions.

The measurement simultaneously of the three dimensions of the movements of points on the chest-wall is obviously a difficult task. It is, however, essential that its difficulty should be overcome—first, in order to obtain an accurate knowledge of these movements; and, second, to learn rightly the action of the different parts of the thorax.

I venture to hope that the problem has been solved by the instrument sketched in the accompanying diagrams. It records on three dials the simultaneous motions of any point on the chest in three planes during one or more acts of respiration.

I propose in the present paper to state the results which I have obtained with this instrument upon both healthy and diseased subjects.

Diagram 1.—Sectional end elevation.

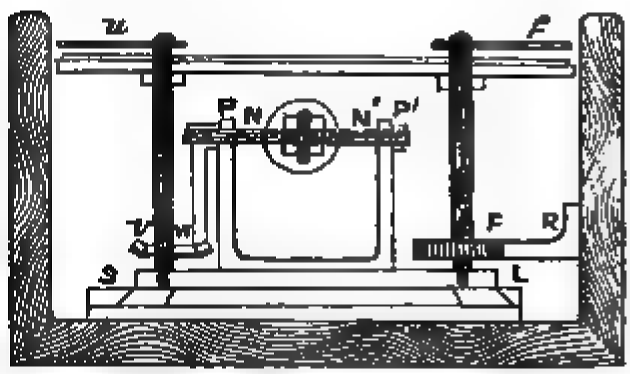


Diagram 2.—Sectional plan.

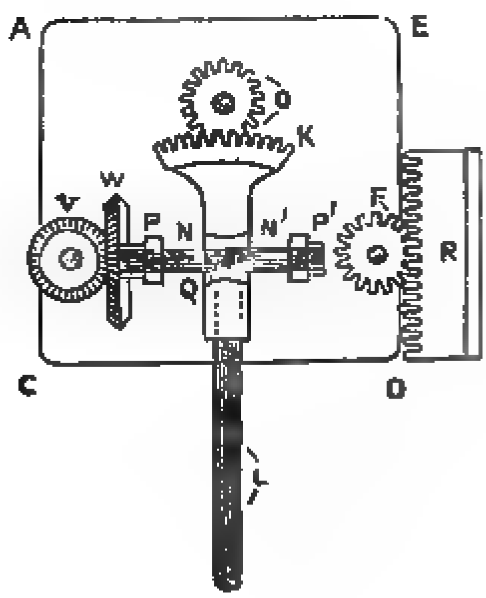


Diagram 3.—Sectional side elevation.

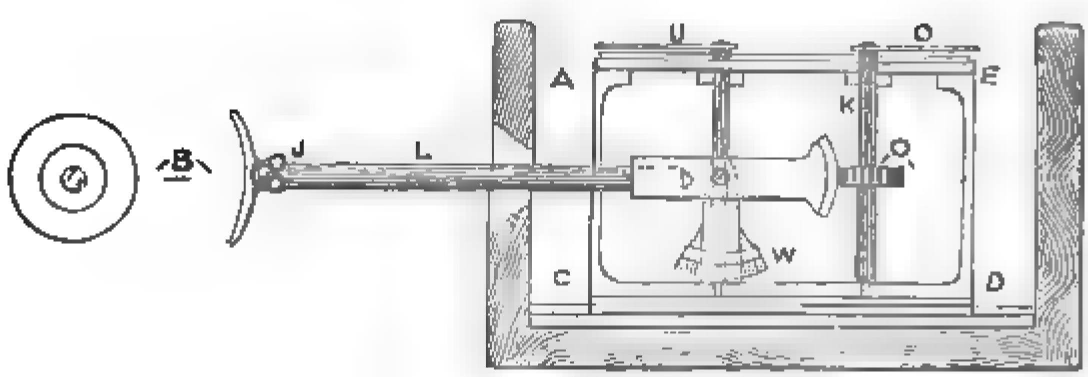
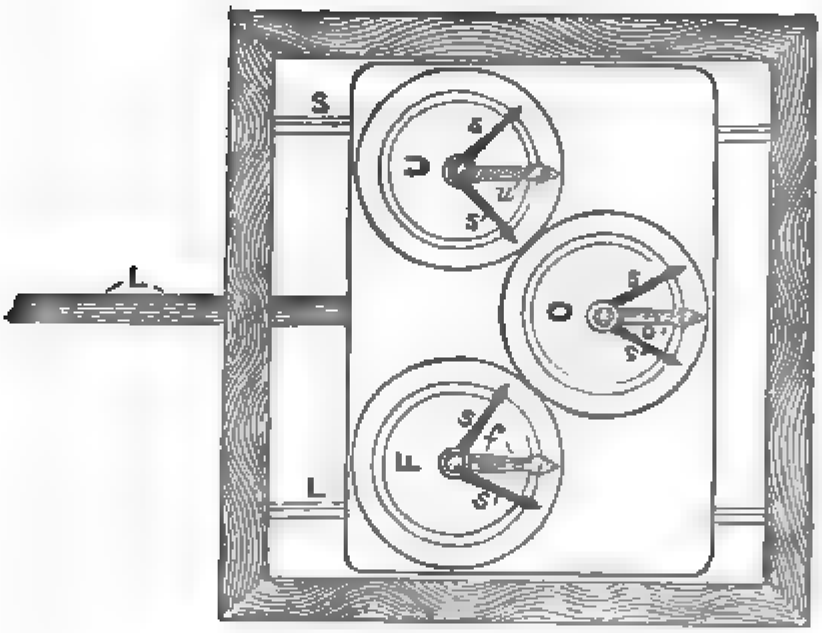


DIAGRAM 4.—Plan.



U. Upwards. F. Forwards. O. Outwards.

1. *Description of the stethometer.*

This stethometer consists of a light steel rod (L),* having at one end a small button (B) attached to it by a ball-and-socket joint (J); the other end of the rod is attached to some simple machinery, which records the motions of the rod, giving duly the movements of the button-end of it forwards, upwards, and outwards.

The forward movement is marked by the sliding forwards of a carriage (figs. 2 and 3, A C D E), carrying a pinion (F), which is turned by a rack (R) placed in the course of the slide (S L), and in its turn this pinion moves an index (figs. 1 and 4, *f*) upon a dial, and the amount of movement is registered by two small loose fingers (fig. 4, *s* and *s'*), which are pushed to each side by the oscillations of the index. The dial is divided into 100 parts, of which each division represents ·01 of an inch.

The upward and downward motion of the button-end of the rod is transmitted through the lever (L) to a shaft (fig. 2,

* See Diagrams 1 to 4.

N N′) placed at right angles to it, which turns upon pivots (fig. 2, P P′) at each end, and upon this again is fastened a segment of a wheel (W, figs. 2 and 3), racked at the side, so as to turn a small pinion (figs. 1 and 2, V) which is attached to the carriage; this pinion turns another index (fig. 4, *u*) which registers the upward motion upon the dial in the same way as index *f*. Every degree of this dial represents ·02 of an inch.

The *outward* motion of the chest is recorded upon a third dial by means of a pinion (O, figs. 2 and 3), also connected with the general carriage.

This pinion is set in motion by the rackwork (figs. 2 and 3, K) attached to the end of the rod (L) beyond the pivot (fig. 2, Q). In order to allow of the upward and downward movements of the end of the rod, this rackwork is made in a peculiar fashion, consisting of vertical divisions of a portion of a sphere, so arranged that only the outward and inward movements of the rod are recorded by the pinion, not those in the upward or downward direction. Every degree on this dial represents ·02 of an inch. It will be evident that by this arrangement of parts each motion is indicated without interference with the others.

The only appreciable error in the indications is to be found in the influence of the radial movements upon the forward motion. This is, however, very easily calculated, and depends upon the length of the lever. Mathematically stated, the distance through which the button moves forwards is registered as less than it really is, by the versed sine of the angle formed by the rod in its angular motion.

The longer the rod or arm, therefore, the less the error from this source. In the model before you it amounts to ·021 of an inch for every 100 degrees of upward or outward motion.

When the instrument is used it is placed in a box and fixed by means of a split ring and screw to a pillar at the required height, and this pillar is furnished with a clamp, by which it is fastened perpendicularly to any convenient table.

Mode of application.

The patient, with the chest exposed and devoid of any ligature, &c., is seated upon a straight-backed chair, which fixes the head and shoulders, and, if possible, his head is also allowed to rest against a support. In this manner the probability of any side movement and any bending of the spine not due to respiration is reduced to a minimum. The button-end of the rod is then brought against any point of the chest, in such a manner that the direction of the rod is perpendicular to the plane of the shoulders, and also perpendicular to the front of the box containing the apparatus.

The distance of the patient from the apparatus also is so arranged that in ordinary breathing the carriage containing the machinery rests about half way upon the slides; in other words, that the index *f* is about its mid position upon the dial. The registering fingers are then applied to each side of the indices, and when it is required to know the extent of forced breathing, the patient is directed to take a deep inspiration and then to make as complete an expiration as possible. The rod is then fixed, and the extent of the movements in the three directions read off upon the dials.

When it is desired to take the extreme extent of several respirations this is done by allowing the patient to go on breathing forcibly several times before the instrument is stopped, and if it is needful to ascertain the extent of movement in ordinary breathing, the patient is permitted to take several quiet respirations before the registering fingers are placed in position.

Sources of error.

From what has been said before, it will have been obvious that there are several sources of fallacy to be regarded in applying the instrument, and corresponding precautions to be taken. In the first place, the mode of

breathing, especially when it is forced, may be unnatural and irregular from nervousness, officiousness, or anxiety; the ribs may be even kept entirely fixed, and the breathing abdominal.

It is necessary, therefore, to urge the patient to breathe easily and naturally, and when a deep inspiration is needed, to ask him to breathe quietly and deeply; and sometimes a little time has to be given, and the attention has to be drawn away from the instrument.

It is not necessary to state that those observations in which the breathing has not been natural were rejected as worthless.

It is also desirable that the muscles of the arms should not be allowed to take part in the process of breathing, as they generally introduce some irregularity in the movements. In all cases, therefore, the patient is directed to rest the tips only of the fingers upon the edge of the table; this arrangement also steadies the body of the patient.

Obesity is a complete bar to the use of the apparatus. If there is much fat upon the ribs it is almost impossible to keep the button of the lever pressed equally upon them; in any case care has to be taken not to move the skin over the bone.

This circumstance is of less consequence, however, since most of the patients who require these observations to be made are already reduced in flesh by disease, and are spare and even thin.

For physiological observations the subjects were purposely selected from such persons.

Points of observation.

It was necessary to fix upon certain points on the chest upon which to apply the button, and most of the observations have been made on the anterior region of the chest. I have also considered it important that the end of the rod should rest on bone; the points selected have, therefore, for the

most part been the top, middle, and lower end of the sternum, the middle of each clavicle, and the middle of each third rib. In males, also, observations were made on the fifth rib, close to the nipples.

Graphical representations of the respiratory movements.

The degree of variation in the different motions may be shown graphically by means of lines drawn to scale as in the annexed diagrams, (diagram 5, &c.), but since one of the

DIAGRAM 5.—Showing the movements of the fifth ribs in a healthy young man, æt. 28.

planes of movement is perpendicular to the other two, the course of one of the three movements must be imagined. The lateral or outward push is, therefore, represented by the device of tracing it at an angle of 45° to the other two.

General movements of breathing.

In ordinary breathing the extent of movement of the ribs is very small, especially in the upper part of the chest, and it is very irregular in extent, even in the same person.

In both men and women a large part of the respiratory

act in ordinary breathing is performed by the diaphragm, a fact remarked by Haller, who says:*—"In naturali inspiratione solum movetur diaphragma, costis ad sensum immotis, nisi quod imæ una vel altera septi motium sequantur."†

Little information, therefore, as to the action of the chest can be obtained by means of any stethometer except in deep or forced respiration.‡

Forced breathing.

When the button of the lever of the above-described stethometer is applied during forced respiration to the front part of the chest, on either side of the sternum, it is observed that a considerable amount of movement is indicated upon the dials, the chief motions being those forward and upward. The forward movement is the more equable of the two, and starts much more rapidly at first than the upward motion. In men the upward movement takes place chiefly at the latter portion of the respiratory act, when the extraordinary muscles of respiration are brought into play.

In most full-chested men the earliest portion of the expansive act seems to be accomplished by an increase of the ordinary action of the diaphragm, then the chest seems gradually to swell outwards, in an ascending order, from below upwards; the ribs are gradually raised by their special

* 'De Musculis Diaphragmatis,' p. 27. Berne, 1733.

† Quod in dormientibus, sanis, tranquillis, planum est. Comp. Winslow, 1165; Galenus, 'De loc. adf.,' l. 4, c. 6; also Hier. Fabricius ab Aquapendente, de Respirationis instrumentis.

‡ Sibson's 'Medical Anatomy,' p. 63. "In the majority of males the thoracic movement during tranquil inspiration is about one twentieth, the abdominal movement one third of an inch. This indicates that the diaphragm descends about half an inch. In the robust the thoracic movement is even less. In two remarkably well-built men, Sewell the American runner, and the third best English runner, it was only one thirtieth of an inch. In many weak persons, on the other hand, it is as much as one tenth of an inch."

muscles, and the lower ribs are probably at the same time straightened at the anterior angle between them and their costal cartilages; at last, for the final effort of inspiration, the head and shoulders are fixed, the spinal column is curved backwards, and all the muscles capable of producing upward movement of the bony cage are exerted to their utmost powers.

In expiration after this effort the operation is reversed. The first to give way seems to be the diaphragm, the abdomen in spare subjects collapses, and then the ribs descend more gradually, beginning first with the upper ribs, and when a forced expiratory effort is required the abdominal muscles are strongly contracted; the ribs, or even the scapulæ, are pressed downwards, and the spine is pushed forward, so as to contract the cavity as much as possible. This seems to be the most frequent order of events so far as I have observed them, but it is important to remark that the action is undoubtedly greatly under the control of the will, and thus variations in the order of movement may be brought about by various causes—habit, disturbing emotion, suggestions from others, or antecedent ideas of what ought to take place. This fact may, perhaps, account for the different descriptions which have been given of the process by different observers.

Order of movement of the ribs.

Haller* considered that the first rib is the fixed point towards which all the others move in succession, the third following the second and so on. Sabatier stated that the ribs differed in the direction of their movement according to their position, the superior ribs moving upwards, the lower ribs downwards, the middle ribs outwards.

Magendie, on the other hand, thought that all the ribs moved at once, and Dr. Sibson seems to share this opinion, whilst Hutchinson and Humphry both take the same view

* De respiratione experimenta anat. Gotting., 1747.

as Haller; thus, Hutchinson says, "The clavicles, shoulders, scapulæ and superior ribs are raised, the sternum advances, the infra-clavicular region swells remarkably upwards and outwards (particularly in females) like a rolling wave, the supra-clavicular region is raised, the whole apex of the thorax is rendered more obtuse, particularly the antero-posterior diameter. The lower ribs at their cartilaginous extremeties spread outwards, increasing both the lateral and antero-posterior diameter of the base of the thorax, the cartilaginous (Gothic) arch formed by the junction of the sixth, seventh, eighth, ninth and tenth ribs below the sternum, becomes more obtuse by their lateral motion, the abdominal space under this arch down to the umbilicus sinks inwards.

"There is an indescribable undulating roll produced by the consecutive action of the respective ribs, which always commences with a superior rib; in costal breathing a lower rib *never* moves first."

Humphry repeats the statement, "In the normal expansion of the thorax," he says,* "the upper ribs are raised first, the others following in quick succession." Dr. Walshe again says that "even in the male the expansile action, if abrupt, commences superiorly." ('On Diseases of the Lungs,' 4th ed. p. 17.)

I do not profess to place my limited experience in opposition to opinions expressed by such able observers, but in working with the stethometer my impression was that the most usual form of breathing, at any rate in males, was the direct opposite of that mentioned by Mr. Hutchinson and Dr. Humphry. I have taken some pains therefore to try to learn which account of the order of movement was most usually correct, and have adopted two methods of testing the matter.

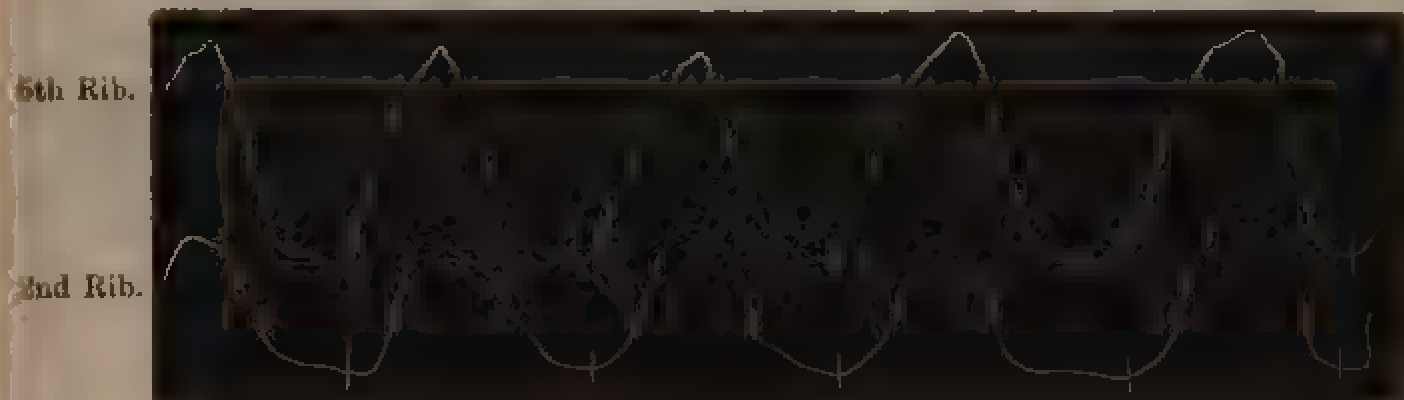
1st. I have fastened tapes of paper around the chest at different heights, whilst the ribs were stationary in the position of somewhat forced expiration, abdominal breathing only being permitted during the process of fastening; slow but deep inspiration was then allowed, and in every instance

* 'Human Skeleton,' p. 349.

which I have so far examined, the order in which the tapes were torn was from below upwards.

2nd. I have had several opportunities of using Dr. Burdon Sanderson's stetho-cardiograph and have applied the button simultaneously to the second and fifth or sixth ribs in both males and females. Diagram 6 gives the tracing obtained in a case to which Dr. Sanderson himself kindly applied it, a young man, in good health and of average size.

DIAGRAM 6.—Simultaneous tracings of the movements of the fifth and second ribs in an adult male, made by the stetho-cardiograph.



The vertical strokes show the points at which the two pens were working simultaneously.

The diameters taken were (*a*) from the fourth vertebra to the second rib, one inch to the right of the sternum; and (*b*) from the eighth vertebra to the fifth rib, half an inch to the right of the sternum. The strokes at the bases of the undulations show the corresponding points of the curves. A comparison of the tracings shows that in every act of breathing, whatever the extent of the inspiration, the lower rib is the first to rise and the last to descend.

It will be noticed that towards the end of the diagram the waves are smaller than at the beginning, showing that respiration was being performed with less effort. The result, however, as far as the action of the ribs is concerned, is the same in every case.

The movements of the second rib are, moreover, less extensive than those of the fifth, as might have been anticipated

from the greater length of the latter bone; but they are also less acute, and more equable in their rise and fall, showing that the work done by the upper ribs is performed more gradually, and that they remain at the point of extreme expansion rather longer than the lower ribs.

This is, I think, what might have been anticipated in the inferior costal type of breathing, since it would need a longer time for the expansion of the upper portions of the lung, if the action had previously commenced in the lower part of the elastic organ—in other words, the inspired air would be partly taken up in expanding the lower part of the lung, and would need a longer time to overcome the elasticity of the upper lobes.

In females, displaying as they do the true "superior costal" type of breathing we might have expected to find that the upper ribs would have a very decided precedence in the order of movement, but this does not seem to have been very decidedly the case in the examples which I have been able to examine by this method.

Several tracings of female respiratory curves were made with the kind assistance of Mr. Hawksley of Blenheim Street, and of these, diagrams 7 and 8 may be taken as examples. The curves show the simultaneous movements of the second and sixth ribs in two healthy young women, during forced breathing, in another instance both ordinary and extraor-

DIAGRAM 7.—Simultaneous tracings of the movement of the second and fifth ribs in an adult female during forced respiration.

DIAGRAM 8.—Tracings of the movements of the second and sixth ribs in an adult female, forced respiration.

dinary acts of breathing were registered. In each case the stays were removed during the experiments.

The positions of the buttons of the stethograph were approximately the same as in the previously given tracing of male respiration.

The peculiar predominence of the action of the upper ribs in female breathing is at once perceived in these diagrams, but it may be observed that in all the curves there is much less difference in the times at which the ribs come into action than in the male chest. As it has been well pointed out by Bérard,* in this type of breathing, the whole of the chest often seems to be raised at once, so that "one sees the clavicles, the sternum, and the first ribs raised, and this action communicated, though in less degree, to the upper part of the inferior portions of the chest." A close examination of the undulations, however, does show a slight difference in the times of rise and fall. A difference, which unlike that observed in male breathing, is sometimes in favour of the upper ribs, sometimes of the lower. In tranquil breathing there was apparent in every act of respiration

* 'Cours de Physiologie,' Paris, p. 258.

a small precedence in the moving of the second rib. In forced respiration the two cases differ on this head, in one (diagram 8) the sixth rib is the first to move and is the longest time in action; and the second rib, although it still has the largest extent of motion, yet lags somewhat behind the other. In the other (diagram 7) the case is reversed, and the second rib retains to a very small extent its precedence. It may also be noticed that for the most part the female respiratory curves are more abrupt in their rise and fall, and hence more pointed than those in the male subject.

Diagram 7 somewhat resembles in its character the tracing of male breathing especially in the more prolonged sustaining of the inspiratory effort by the upper ribs; but even in this tracing there is more abruptness in the general action of both sets of ribs.

It would not be just to assume that these few examples are sufficient to decide this question, but they may serve to show the value of this method of inquiry. It may be observed also that the subject is not without practical importance. There is still some controversy as to whether pulmonary phthisis appears most readily or lasts longer in the more or the less used portions of the lungs, and when definite results have been obtained as to the relative movements of the ribs they may probably be advantageously compared with other statistics relating to this disease.

Ratio of forward and upward movements.

Another point in the description which I have given of the general movements of breathing should also be fully explained since its accuracy might be called in question.

It is stated in the description of the general movement of breathing, that in deep inspiration the stethometer shows a difference in the rates of movement, forward and upward, in the early and late periods of that act. Now since the movements of any point on the chest are, to a great extent, determined by the radial motions of a chord line between that point and the vertebral articulations of the underlying rib, it might appear that there was an error in the observa-

tions, and that the forward motion must necessarily be accompanied by an upward or an outward movement in some definite degree, and, except when the rib is nearly horizontal, there can be little or no upward motion without a simultaneous and commensurate forward or outward movement.

This question is one of much importance and touches upon a very interesting problem relating to the causes of the respiratory movements, which must be fully considered hereafter; but, in the mean time, it may be sufficient to remark that the action of the stethometer gives a strictly accurate representation of the movement, and that the discrepancy noticed is partly due to complex actions of the mechanism of respiration, partly to a simple geometrical law by which the versed sines and cosines of angles are governed.

The movements of the chest in forced breathing.

The regions which have been examined in the greatest number of subjects were, 1st, the sternum at its highest point, its middle, and just above the ensiform cartilage; 2nd, the clavicles near their centres; 3rd, the anterior ends of the third ribs, near their articulations to the cartilages; 4th, the fifth ribs near the nipples.

The actual extent of these movements varies in different individuals and in different regions of the chest; probably no two persons breathe with the same kind or extent of motion. It becomes necessary, therefore, to examine the details of each individual case, and then to attempt to extract from them some general conclusions which represent what is usually found to exist. A fair representative selection of examples must thus first be given in order to illustrate the results obtained with the three-plane stethemoter.

The following table records the motions of different parts of the chest in healthy adult males, who were purposely selected as men of average capacity in breathing. No examples of extraordinary power are given, as they would be less suitable to the objects of the inquiry.

TABLE 1.—*Extent and direction of the movement of different points on the chest in 100ths of an inch.*

Extent and direction of movement.	Top of sternum.	Middle of sternum.	Ensiform cartilage.	Centre of clavicle (right).	Centre of clavicle (left).	3rd rib (right).	3rd rib (left).	5th rib (right).	5th rib (left).	Remarks.
1. Forwards	90	96	80	90	78	80	85	102	102	Male, æt. 35, healthy.
Upwards	111	120	99	108	105	129	135	135	135	
Outwards	.	...	..	.	...	27	27	30	30	
2. Forwards	71	90	127	.		119	94	144	122	Male, 40, tall, healthy.
Upwards	75	90	114	...	...	115	135	150	135	
Outwards	.	..	..	...	.	35	40	45	45	
3. Forwards	107	114	85	60	54	92	75	95	85	Male, æt. 30, tall, healthy.
Upwards	120	120	105	75	75	75	75	75	105	
Outwards	..	..	...	...	...	30	36	40	36	
4. Forwards	..	98	...	56	59	124	127	112	102	Male, æt. 28, healthy, strong.
Upwards	...	111	..	99	90	150	165	180	180	
Outwards	.	...	..	...	..	24	30	36	30	
5. Forwards	85	85		59	59	...	...	...	.	Male, æt. 45, middle height.
Upwards	135	150	...	135	135	..	...	...	..	
Outwards	..	.	..	...	..	..	.	..	.	

6. Forwards	...	75	25	25	25	35	30	...	...	Male, æt. 40, tall, rather stout.
Upwards	...	60	50	60	60	50	60	...	...	
Outwards	...	...	...	...	...	10	10	...	...	
7. Forwards	55	50	50	40	45	50	65	85	90	Male, æt. 23, slight, healthy, left-handed.
Upwards	110	110	120	100	110	110	120	160	160	
Outwards	...	...	...	...	...	15	15	25	30	
8. Forwards	85	93	95	80	85	135	115	115	110	Male, æt. 32, tall, healthy, muscular.
Upwards	100	120	115	130	130	160	150	110	120	
Outwards	...	...	...	...	...	30	25	30	30	
9. Forwards	25	55	100	55	50	85	78	100	100	Male, æt. 42, 6 ft., healthy, active.
Upwards	50	110	100	100	90	130	115	130	110	
Outwards	...	...	...	...	...	15	12	20	15	
10. Forwards	25	35	40	25	25	50	50	50	50	Male, æt. 21, slight, healthy, middle height.
Upwards	20	35	40	30	30	35	35	50	40	
Outwards	...	...	...	...	...	10	10	12	14	
11. Forwards	30	25	43	25	23	...	...	68	50	Male, æt. 32, 6 ft. high, healthy, strong.
Upwards	65	60	65	60	60	...	...	84	90	
Outwards	...	...	...	...	...	...	...	20	25	
12. Forwards	15	30	45	25	20	70	60	55	65	Male, æt. 22, 5 ft. 6 in., healthy, athletic.
Upwards	30	85	100	40	35	120	100	100	100	
Outwards	...	...	...	...	...	15	14	20	20	
13. Forwards	68	85	...	50	50	105	105	...	...	Male, æt. 45, 5 ft. 8 in., healthy, slight.
Upwards	65	100	...	60	54	95	100	...	...	
Outwards	...	...	...	...	...	10	15	...	...	

Regional movements.

The sternum.—It might be supposed that, in consequence of the mode of its attachment to the ribs and clavicles, the sternum would only be able to move in the upward and forward directions, and that it could not deviate from the middle line of the body, but owing to the different size of the two halves of the thorax, and to the varying strength of the muscles on each side, it is usual even in healthy persons to find a slight difference in the degree of motion of the right and left ribs, either in the forward and upward or outward directions; this fact must needs cause some slight lateral movement of the sternum, especially at its lower end, which would be drawn towards that side which has the least power of expansion.

It would, however, require great care in fixing the body in order to note the very small deviations which would arise from this source in most individuals, and the readiness with which this outward reading of the instrument is affected by movements of the body renders it untrustworthy for very small readings. It is on this account that the record of this motion is sometimes omitted from the tables even in the measurement of the third or fifth ribs.

In disease or malformation of the thorax, either congenital or accidental, the lateral movement of the sternum may, however, become very perceptible; the following table gives the movements of the sternum in three cases.

TABLE 2.—*Movements of the sternum.*

Movements of the sternum.	Forward.	Upward.	Outward.
(1). Male, æt. 21. Tubercle; contracted pleuritic adhesions on left side . .	85	45	12 (L.)
(2). Female, æt. 48. Spinal curvature to right side	35	60	8 (L.)
(3). Female, æt. 33. Crushed ribs on the right side; upper sternum . .	25	20	12 (R.)
(4). Same case. Lower sternum . . .	55	100	15 (R.)

As a general rule, the sternum moves forward and sometimes upward more freely than the clavicles, and the motions of the lower portions of the bone are greater than those of its upper end. There is thus an alteration of its plane towards a less vertical position, but this tilting forward of the lower sternum is not as great as might have been concluded from the greater length of the lower ribs, their greater mobility, and the obliquity of the costal cartilages. If the problem were purely a mathematical one, there ought to be some definite relation between these conditions and the extent of motion of the sternum, but it cannot be traced in the record of measurements given in the tables. The explanation of this fact is probably to be found in the yielding nature of the levers employed, and in the action of the great inspiratory muscle, the diaphragm. The action of this muscle on the six lower ribs will probably prevent the forward and upward motion of the sternum and, perhaps, also of the ensiform cartilage in children. To some extent it would appear from the tables that the more elastic the thorax, the more is this influence of the diaphragm felt.

Many of the remarkable vivisections made by Traube would seem to bear out this explanation of the lesser degree of motion of the lower end of the sternum.

For example, at p. 150 ('Gesammelte Beiträge') he says, "The only influence which, according to our experience, the diaphragm exerts upon the lower part of the thorax, consists in the drawing inward of the foremost (bony) ends of the seventh and eighth or from the seventh to the ninth rib." This opinion is fully borne out by his experiments, notably the tenth, eleventh, and the seventeenth.*

The clavicles show, as might have been predicted, more upward than forward movement, and they are especially affected by the final effort of breathing, when the extraordinary muscles attached to them are brought into action.

There is little or no outward motion of any point on these bones, or on the first and second ribs in the anterior region

* See also Mr. Le Gros Clark's "Remarks on the Mechanism of Respiration," 'Proc. of Roy. Soc.,' vol. xx, p. 122.

of the chest, but on the third rib this motion is already distinct, and it increases in amount as we descend to the lower ribs, where the costal cartilages become more and more oblique.

The ribs at their anterior ends generally move more decidedly upward than the sternum, owing, perhaps, to their power of outward rotation, which permits them to rise more freely. Their forward push is also very considerable, especially in the lower ribs, the fifth and seventh, and in some cases even the second and third ribs enjoy more forward movement than the sternum.

In most healthy male subjects the fifth and seventh ribs enjoy much more general mobility than the third, but this is by no means universally true, and possibly in obese persons this might not be even the usual case, since in them the so-called superior costal type of breathing may prevail. I have not, however, been able to apply my instrument in these cases.

There is certainly no regular increment of movement in the ribs as we descend from the upper to the lower. Owing to the gradual increase in the length of the ribs from the first to the eighth or ninth, we might have expected that this circumstance would have given them a wider sweep at their anterior ends, but I can fully corroborate Dr. Sibson's statement that in some cases the second or third ribs have more power of forward movement than the fifth or even the eighth. There is also in the tables abundant evidence of the independent action of the different ribs. Mr. Hutchinson has well pointed out this power of independent movement in the case of certain chest diseases, and in most of the cases of the table it will be found that the third and fifth ribs are by no means in exact accord in the proportion which the several dimensions of the movements bear to one another.

The outward movement of points upon the five or six upper ribs appears to increase as they recede from the sternum until a limit is reached about midway between that bone and the vertebral column, beyond this point, measuring from behind, this motion naturally again gradually diminishes.

TABLE III.—*Dimensions of the movements of respiration in 100ths of an inch. Adult females.*

Extent and direction of movement.	Top of sternum.	Middle of sternum.	Ensiform cartilage.	Centre of clavicle (right).	Centre of clavicle (left).	3rd rib (right).	3rd rib (left).	Remarks.
1. Forwards	51	...	...	51	51	61	59	J. H., female, æt. 27, healthy (stays on).
Upwards	45	...	...	51	60	60	63	
2. Forwards	117	...	...	85	102	...	...	E. R., female, æt. 27, healthy (wearing stays).
Upwards	114	...	...	99	99	...	...	
3. Forwards	76	102	96	60	85	119	105	L. E., female, æt. 29, healthy (no stays).
Upwards	75	90	90	90	90	105	105	
Outwards	...	...	...	...	...	30	30	
4. Forwards	51	85	93	51	60	60	76	M. F., female, æt. 22, healthy (no stays).
Upwards	54	51	45	60	75	96	96	
Outwards	...	...	...	...	...	30	33	
5. Forwards	31	51	...	42	46	68	76	M. D., female, æt. 33, healthy (no stays).
Upwards	105	94	...	60	75	99	105	
Outwards	...	...	...	...	...	...	...	
6. Forwards	87	40	...	42	38	51	51	B. M., female, æt. 36, healthy (no stays).
Upwards	70	75	...	75	75	90	105	
Outwards	...	...	...	...	...	21	18	
7. Forwards	30	35	...	50	55	75	90	M. R., female, æt. 34, healthy (no stays).
Upwards	50	60	...	70	75	110	120	

Variations in healthy breathing.

1. *Influence of sex.*—The preceding table gives a few of the results which have been obtained by means of the three-plane stethometer in young and healthy women.

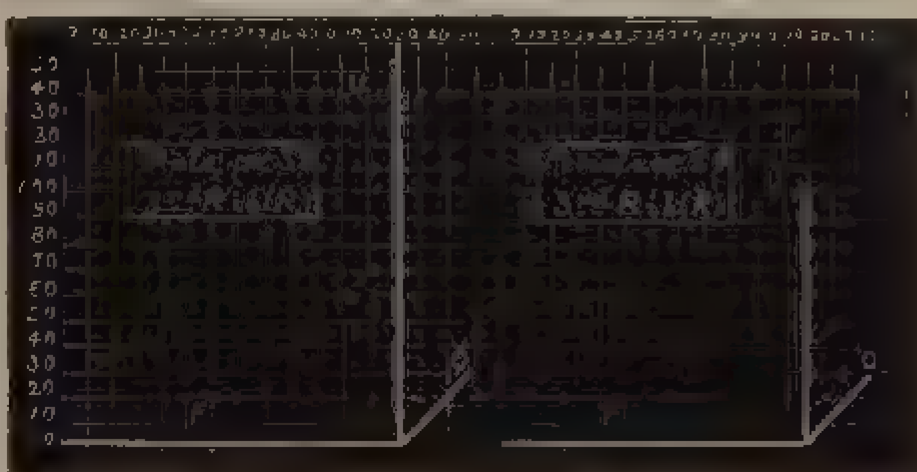
It has been noticed by most observers that in women both the ordinary and extraordinary breathing is chiefly costal; and since this fact has not been successfully traced to the fashion of dress, it seems to be probably arranged to facilitate gestation.*

It does not appear to have been remarked, however, that the superior costal type of breathing does not give proportionately any increased power of raising the ribs in forced inspiration.

In the cases which I have examined it was rare to find any female who could raise the clavicles and third ribs more than one inch, and if the proportions between the motions are altered at all it is rather to the advantage of the forward thrust, which in several instances attained to or exceeded one inch.

Diagram 9 gives a representation of the movements of the third rib in a healthy male and female.

DIAGRAM 9.—Dimensions of movements in males and females.



* This opinion was held by Boerhaave and Haller, but, as Dr. Walshe points out, if this were true ascitic females ought to escape dyspnœa, and the difference between male and female breathing is much less during sleep, and male and female animals breathe almost exactly alike.—'Diseases of the Lungs,' Ed. 4, p. 16.

The peculiarities of motion of the different bones are also for the most part similar in females and males.

The forward movement of the middle of the sternum slightly exceeds in most cases that of the manubrium, and again we find the total movement of the ensiform cartilage somewhat lessened. The clavicles move at about the same rate as the sternum, in most cases rather less, in some rather more than that bone, and they move considerably less than the third rib, which as in men moves more than either sternum or clavicles.

The fifth ribs were not examined. It is somewhat remarkable that, in most of the women examined, the motions on the left side exceeded those of the right.

2. *Age.*—The following table contains the measurements of the respiratory movements in children and old persons of both sexes :—

NOTE.—The measurements recorded in the following tables were obtained before I became acquainted with Dr. Sibson's observations on this subject. It is, however, interesting to notice that his records of the forward motion of the several parts correspond very closely with those which I have obtained, and his deductions from the figures are often strikingly similar to those which I have ventured to draw. (See Table VII (*a*), on page 91.)

TABLE IV.—*Dimensions of the movements of respiration in* 100*ths of an inch, in young children and old people.*

Direction of movement.	Top of sternum.	Middle of sternum.	Ensiform cartilage.	Clavicle (right).	Clavicle (left).	3rd rib (right).	3rd rib (left).	5th rib (right).	5th rib (left).	Remarks.
1. Forward	98	98	...	64	64	110	102	110	102	Male, æt. 11, healthy, tall.
Upward	111	96	...	117	120	75	100	100	100	
Outward	...	...	...	...	...	20	20	30	25	
2. Forward	...	...	...	30	29	...	...	...	...	Female, æt. 6, healthy, small.
Upward	...	...	...	36	36	...	...	...	...	
3. Forward	45	...	...	55	55	95	85	...	...	Male, æt. 9, healthy, strong.
Upward	50	...	...	110	110	120	120	...	...	
Outward	...	...	...	...	...	30	30	...	...	
4. Forward	17	25	30	45	40	35	30	30	35	Male, æt. 80, gardener, healthy.
Upward	45	50	54	50	50	45	55	50	55	
Outward	...	...	...	...	...	10	10	10	10	
5. Forward	30	20	10	20	...	25	35	25	25	Male, æt. 59, merchant, healthy.
Upward	100	75	60	100	...	100	100	70	75	
Outward	...	...	...	...	...	15	15	20	20	
6. Forward	10	25	25	15	15	30	35	30	30	Male, æt. 66, healthy, spare.
Upward	50	30	30	45	35	50	70	55	60	
Outward	...	...	...	...	...	...	...	...	...	
7. Forward	45	35	...	30	30	50	45	...	...	Male, æt. 70, healthy.
Upward	70	70	...	50	35	90	100	...	...	
Outward	...	...	...	...	...	20	20	...	...	

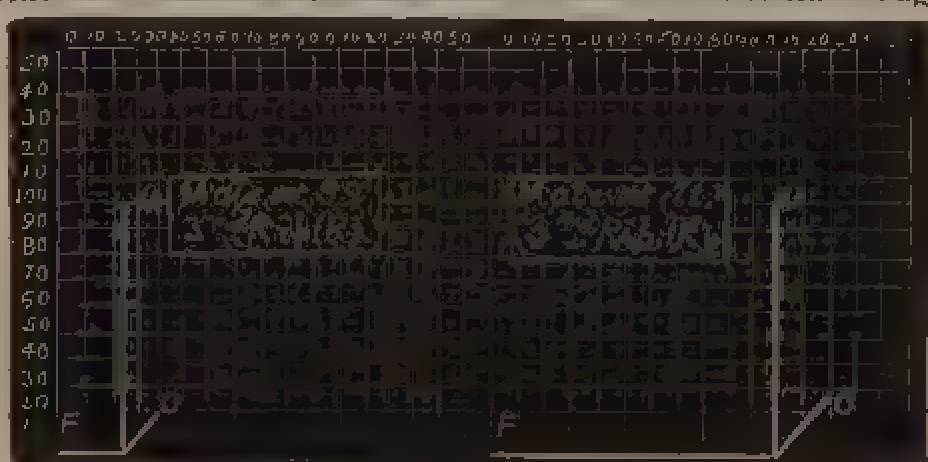
It is interesting to observe the large relative movement of the chest-walls in children as compared with that of adults, and to notice the diminution in the power of chest motion as age advances.

This fact arises partly, no doubt, from the greater mobility of the bones, and elasticity of both bones and cartilages in youth; partly, perhaps, from the shape of the thorax in children, the deep lateral grooves on each side of the spine being absent; there is thus less difference between the chord lengths of the ribs of children and those of adults than might be anticipated, and the chest in children being shorter from above downwards, all the movements forward and upward are necessarily exaggerated in order to produce a corresponding alteration in the capacity of the chest. It is possible also that muscular energy in children may be comparatively greater in proportion to the amount of resistance in the respiratory muscles.

I have not observed that the proportions between the three dimensions of the movements are very different in children and adults, especially female adults, except that the forward push is somewhat increased. In old age, however, there is both a general diminution in all the dimensions of the movement, and in some cases the forward motion is lessened to the lowest degree compatible with any angular rise of the ribs.

The following diagram affords a means of comparing the movements of the third ribs in a child of eleven and a man of fifty-nine; the difference in the degree of forward movement is very striking:—

DIAGRAM 10.—Showing dimensions of movements in youth and age.



3. *Position.*—It is scarcely necessary to point out that position has much to do with the freedom of breathing, and interesting observations on this subject have been made by Drs. Sibson and Hutchinson. I have myself, as yet, only made observations upon the influence of the position of the arms upon the breathing of persons in a sitting posture, especially as to the relative motions, upward and forward, of the sternum.

The following tables show the variations in these movements in three individuals :—

TABLE V.—*Movements of the centre of the sternum in different positions of the arms.*

	Case I.		Case II.		Case III.	
	F.	U.	F.	U.	F.	U.
a. Hands supported on a level with the shoulders or on the table	102	105	107	114	90	100
b. Hands resting on the sternum	93	99	92	84	90	90
c. Hands locked behind the head	90	70	43	70	75	60

TABLE VI.—*Showing the movements of the lower end of the sternum in different positions of the arms.*

	Case I.		Case II.		Case III.	
	F.	U.	F.	U.	F.	U.
a. Hands supported on a level with the shoulders or on the table	76	115	115	150	100	120
b. Hands resting on the chest	73	84	105	129	95	115
c. Hands locked behind the head	65	66	98	129	...	...

The movements most affected were those forward, but both the upward and forward motions were impeded by any position of the arms, except that on a table or on a level with the shoulders. This observation shows, therefore, that the well-known position of the asthmatic patient during a paroxysm of dyspnœa is the one which really gives the greatest amount of power of moving the chest-walls.

4. *Muscular power.*—In a very large proportion of the male cases examined it was found that the movements on the right side of the chest exceeded those on the left—a fact which may be placed in relation to the observations of different observers, notably Drs. Sibson, Wintrich, and Walshe, that in right-handed men the circumference of the right side may naturally exceed that of the left by from one half to three centimetres. But it appears probable that both the increased power of movement and the increased size on the right side are due to the greater force of the muscles on this side. The power of movement certainly is remarkably affected by the muscular force of the subjects, and it is reasonable to believe that the increased size of the right side of the chest may be, partly at least, due to the same cause. The cases in which the greatest amount of movement of the ribs and sternum was observed were almost always men of considerable muscular development, although, even in these men, those who were most lithe and active, and who therefore had the greatest freedom in their joints, could exceed in some of their chest measurements the more sturdy and more muscularly strong.

Variations in the Respiratory Movements in Disease.

The following table gives the dimensions of the respiratory movements in phthisis and other lung diseases.

TABLE VII.—*Dimensions of the movements of respiration in 100ths of an inch in phthisis—males.*

Extent and direction of movement.	Top of sternum.	Middle of sternum.	Lower end of sternum.	Clavicle (right).	Clavicle (left).	3rd rib (right).	3rd rib (left).	5th rib (right).	5th rib (left).	Remarks.
1. Forward	51	...	...	48	39	67	42	...	...	Male, æt. 50, right, large incontracted vomica, dry; left, recent tubercle.
Upward	45	...	...	45	51	66	51	...	...	
Outward	...	...	...	...	...	30	27	...	...	
2. Forward	...	...	...	54	44	...	...	...	...	Male, æt. 18, incipient phthisis, on left side.
Upward	...	...	...	75	63	...	...	...	...	
3. Forward	92	...	...	...	...	80	85	...	...	Male, æt. 26, right, first stage of phthisis.
Upward	90	...	...	...	...	90	105	...	...	
Outward	...	...	...	...	...	24	36	...	...	
4. Forward	42	...	...	34	54	93	85	...	...	Male, æt. 60, slight, incipient tubercle on right side.
Upward	69	...	...	69	57	69	105	...	...	
Outward	...	...	...	...	...	...	...	...	...	
5. Forward	...	...	...	...	...	68	59	...	...	Male, æt. 24, side tubercle softening on left side.
Upward	...	...	...	...	...	70	69	...	...	
6. Forward	24	34	34	...	...	59	41	105	51	Male, æt. 25, incipient deposit on left side, not
Upward	39	36	35	...	...	60	45	45	36	
Outward	...	...	...	...	...	30	15	30	20	
7. Forward	46	...	...	24	24	39	42	...	...	Male, æt. 32, small weak chest, deficiency of breathing left side.
Upward	60	...	...	33	39	60	48	...	...	
Outward	...	...	...	...	...	20	15	...	...	
8. Forward	56	85	...	85	54	110	68	110	68	Male, æt. 21, tubercle both sides, formerly pleurisy on left, and contraction.
Upward	39	45	...	41	39	75	39	75	39	
Outward	...	12	...	...	...	...	...	...	...	
9. Forward	50	55	55	55	55	48	55	60	45	Male, æt. 21, tubercle under right clavicle.
Outward	80	80	80	90	90	60	90	60	50	

10. Forward............	24	34	34	...	...	59	41	105	51	Male, æt. 26, chronic phthisis on left side, extending five or six inches.
Upward	39	36	45	...	...	60	45	45	36	
Outward............	...	...	...	...	...	20	15	20	15	
11. Forward............	25	30	...	50	15	100	70	70	45	Male, æt. 29, chronic phthisis on left side.
Upward	45	40	...	50	25	60	60	60	40	
Outward............	...	...	...	...	...	10	12	25	10	
12. Forward............	25	35	30	25	20	55	35	50	40	Male, æt. 57, chronic phthisis on left side, first stage.
Upward	65	60	60	70	60	70	70	60	60	
Outward............	...	...	...	...	...	15	10	20	15	

Table VII (*a*). [*See note, page* 85.]

The following table gives a few of Dr. Sibson's measurements in healthy subjects in forced breathing:

Case.	Age.	Sternum.		2nd rib.		4th and 5th ribs.		6th rib.		8th rib.		10th rib.		Remarks.
		Upper.	Lower.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	
1.—Adult Males.														
4	23	120	75	130	125	120	120	120	120	...	...	...	...	
8	28	100	120	115	115	120	120	130	130	130	130	80	80	
9	37	130	110	110	100	95	90	80	80	95	90	95	80	
12	26	180	190	225	...	...	...	...	...	...	...	...	...	
13	26	100	100	150	...	...	...	...	...	...	...	...	...	
16	22	90	60	100	100	100	100	50	40	...	...	...	...	
2.—Boys.														
18	16	50	110	90	70	60	90	...	...	100	75	60	45	
22	15	60	70	100	80	...	...	100	95	90	75	80	70	
25	11	110	50	130	130	90	85	40	35	60	60	35	25	
26	10	70	50	100	100	100	100	...	...	50	50	...	...	
3.—Old Men.														
39	61	90	90	75	70	80	80	60	60	60	60	22	22	
40	68	100	80	50	50	75	75	50	50	30	25	...	...	
41	65	80	90	50	40	50	45	73	35	30	30	...	...	
42	71	50	50	55	55	35	20	20	20	10	50	2	2	

TABLE VIII.—*Dimensions of movements in phthisis—females.*

Extent and direction of movement.	Upper sternum.	Middle sternum.	Lower sternum.	Clavicle (right).	Clavicle (left).	3rd rib (right).	3rd rib (left).	Remarks.
1. Forward	30	40	...	35	45	65	50	Female, slight tubercular congestion under right clavicle.
Upward	50	50	...	40	53	70	50	
Outward	...	...	...	...	...	10	7	
2. Forward	49	...	...	47	39	68	56	Female, F. D., æt. 19, right side healthy; left, softening tubercle.
Upward	57	...	...	75	36	63	45	
Outward	...	...	...	...	...	36	21	
3. Forward	34	...	...	17	27	42	42	Female, W. B., æt. 48, incipient tubercular deposit under right clavicle.
Upward	60	...	...	60	24	60	51	
4. Forward	56	...	...	46	39	59	59	Female, æt. 47, tubercle on both sides, softening on left.
Upward	39	...	...	45	45	48	36	
5. Forward	39	42	56	19	37	42	68	Female, æt. 37, right side, extensive tubercular deposit, softening; left, incipient tubercle.
Upward	60	60	60	45	54	60	81	
6. Forward	42	42	34	...	...	54	41	Female, æt. 16, left side, incipient deposit.
Upward	45	45	45	...	...	45	39	
7. Forward	25	30	...	30	25	35	35	Female, æt. 38, left side, incipient deposit.
Upward	50	50	...	60	60	75	75	

8. Forward............	15	35	...	30	12	35	30	Female, æt. 32, left side, softening tubercle advanced.
Upward	50	60	...	40	40	70	40	
Outward............	...	...	...	...	...	12	4	
9. Forward............	...	45	...	25	20	65	50	Female, æt. 23, incipient tubercle on left side.
Upward	...	60	...	50	55	80	70	
Outward............	...	...	...	...	...	...	...	
10. Forward............	25	55	...	58	28	60	75	Female, æt. 23, chronic tubercular deposit under left clavicle.
Upward	70	80	...	90	70	100	100	
Outward............	...	...	...	...	...	20	20	
11. Forward............	15	35	...	30	12	35	30	Female, æt. 32, advanced phthisis on both sides.
Upward	25	30	...	20	20	35	20	
Outward............	...	...	...	...	...	6	2	
12. Forward............	30	30	...	30	25	40	35	Female, æt. 20, chronic phthisis both sides, large dry vomica on left.
Upward	20	15	...	25	20	30	52	
Outward............	...	...	...	...	...	...	...	
13. Forward............	15	10	...	10	15	16	22	Female, æt. 25, advanced phthisis both sides.
Upward	50	40	...	24	50	55	55	

TABLE IX.—*Dimensions of movements in other chest affections.*

Extent and direction of movement.	Top of sternum.	Middle of sternum.	Lower end of sternum.	Clavicle (right).	Clavicle (left).	3rd rib (right).	3rd rib (left).	5th rib (right).	5th rib (left).	Remarks.
1. Forward	17	19	...	17	17	20	34	3	20	Male, æt. 30, advanced emphysema and chronic bronchitis.
Upward	36	45	...	45	36	45	45	21	30	
2. Forward	28	...	21	...	...	42	34	...	...	Male, æt. 50, chronic bronchitis, incipient tubercle on left side.
Upward	51	...	51	...	...	60	45	...	...	
Outward	...	...	...	...	...	20	15	...	...	
3. Forward	56	...	...	46	39	59	59	...	...	Female, æt. 47, chronic bronchitis, slight tubercle on both sides.
Upward	39	...	...	45	45	48	36	...	...	
4. Forward	5	25	...	...	...	25	25	20	20	Male, æt. 50, chronic bronchitis, with asthma.
Upward	20	40	...	...	...	40	38	30	35	
5. Forward	...	...	...	39	34	42	41	...	...	Male, æt. 50, emphysema and chronic bronchitis.
Upward	...	...	...	57	36	57	54	...	...	
6. Forward	...	35	35	...	...	30	25	25	40	Male, æt. 65, chronic bronchitis (incomplicated).
Upward	...	57	40	...	...	50	50	35	50	
7. Forward	39	...	...	68	29	54	39	51	25	Male, æt. 19, pleurisy left side.
Upward	75	...	...	80	66	99	84	135	60	
8. Forward	42	...	...	68	34	42	59	34	34	Same case after two months, pleurisy on the right side having intervened.
Upward	150	...	...	135	165	150	180	180	210	

9. Forward............	35	55	...	50	95	50	67	20	75	Male, æt. 13, pneumonia and pleurisy on right ride.
Upward	45	50	...	40	100	40	70	40	100	
Outward............	...	...	...	...	...	7	8	...	...	
10. Forward............	25	35	...	15	20	32	50	...	...	Female, æt. 48, spinal curvature to right side.
Upward	40	60	...	30	50	41	64	...	...	
Outward............	...	8 (L)	...	...	...	6	8	...	...	
11. Forward............	25	55	...	15	18	55	55	...	...	Female, æt. 25, ribs on right side crushed severely.
Upward	20	100	...	25	20	100	100	...	...	
Outward............	12	15	...	...	...	6	12	...	...	
								2nd rib. R.	2nd rib. L.	
12. Forward............	25	87	...	30	25	75	110	72	80	Male, æt. 48, with syphilitic nodes on 2nd and 3rd right ribs.
Upward	55	82	...	45	40	90	100	60	80	
Outward............	...	...	...	...	...	...	...	...	...	
13. Forward............	25	35	...	18	18	45	40	...	...	Male, æt. 21, pleurisy on both sides, commencing tubercle on left.
Upward	25	40	...	25	20	80	40	...	...	
Outward............	...	...	...	...	...	15	3	...	...	

The cases of disease are thirty-eight in number, not sufficient [illegible] modifications of movement caused by the various lung diseases; but since the number of separate measurements recorded in the tables amounts on the whole to nearly [illegible], they may, perhaps, afford data for remarks upon the results—at least, so far as they bear upon the physiology of respiratory movement.

A survey of all the cases on the register shows, as might have been expected, that the movements of respiration in disease are, on the whole, much less extensive than those in health. It is difficult at first glance to see how disease can ever produce exaggeration of motion except by bringing about an enlargement of the sound side to compensate for the loss of breathing on the other. The extent of motion of the bony walls also in the ordinary breathing of some diseased subjects may, by habit, be increased, the voluntary muscles being more brought into action; but, even in this case, the limits of extreme expiration and inspiration will probably be less than in health.

Increased movement may, however, in rare cases, be brought about by disease, especially in the extent of the upward motion.*

Cases 7 and 8 (Diagram 11), affords a good instance of this fact. From this patient two series of measurements were taken, the first soon after the pleuritic effusion on the left side had taken place; the second about two months afterwards, another attack of pleurisy with effusion on the right side having intervened.

It is very interesting to note the great increase in the upward readings in this last series of observations, due, no doubt, to an increased use of the respiratory muscles, which seem to have become stronger by use; at the same time it may be remarked that in the lower regions of the chest the forward motion is reduced to the smallest dimension compatible with the degree of upward motion recorded.†

* Dr. Walshe says, the chest movements may "exceed the health[illegible] where a muscular effort is made to overcome some obst[illegible] chest."

† If the initial angle of the rib be taken to have bee[illegible]

DIAGRAM 11

Case[illegible] tients are
also p[illegible] motion to
com[illegible] No. 4 it is the
[illegible] to be attempting
[illegible] the injured portion of
[illegible] the forward di[illegible] ch
[illegible]

is increased out of all ordinary proportion to the upward rise.

In No. 9 perhaps both the forward and upward movements of the right fifth rib are increased to take the place of the deficient working of the right third rib.

Amongst females the examples of this compensatory increase of motion are less frequent; perhaps because the action of the fifth ribs could not in them be examined. Nos. 1, 3, and 10, however, show an increase; in case No. 3 of the upward, in the latter in the forward, motion of the third rib on the affected side. In No. 1 both forward and upward motions are increased in the third rib on the diseased side.

Diminished movement.

It is amongst emphysematous and asthmatic patients that the most distinct examples of extreme diminution of motion are to be found, and in these cases all the three dimensions of the movement seem to be equally lessened. Diagram 12 represents the movements of the third rib in a case of advanced emphysema.

DIAGRAM 12. Case of emphysema, æt. 30.

The rigidity of the thorax in these disorders seems to offer an almost equal impediment to all the movements of the ends of the ribs.

In chronic bronchitis there is also usually to be found considerable diminution of motion.

An observation from which it would appear that even in this disease there must be in most cases a loss of elasticity, and in many, probably, some emphysema as well. It might otherwise have been anticipated that the respiratory muscles in these patients might at times have increased in power, and thus might have given greater extent of forced breathing.

There can be no doubt, however, that in all these cases there is exaggerated costal motion in ordinary enforced respiration.

In phthisis cases are recorded in which both lungs were affected, and others in which only one lung was as yet attacked. For the purpose of comparison with the healthy movements, the latter class was most valuable; but some use may be made of the other records to show the effect of softening or of the formation of vomicæ, as compared with that of the incipient deposition of tubercle.

In both males and females it was found that one of the earliest indications of the disease was a diminution of movement over the part immediately affected, and here again it is usually the forward indication which is most retarded.

In most cases the proportion of this dimension to the upward rise is more or less diminished.

In some the upward dimension is unaltered, and in others it is apparently increased.

In three cases amongst the males (Nos. 1, 8, and 10) and four cases among the females (Nos. 1, 2, 4, 8), the ratio of the forward to the upward movement was somewhat in excess on the side on which the disease was most advanced; but it is a significant fact that in five of these cases softening had taken place to a considerable extent, and in 3 (M. 1, 8, F. 4), not only had vomicæ formed, but they had dried up, and were for the time quiescent.

It may be remarked also, that these cases were of very long standing, and that they seemed to have a tendency towards a more or less complete recovery, notwithstanding the fact that these patients had exhibited at one period of this malady excessively severe symptoms.

Cases (males) 4 (females) 3, 7, show plainly the mode in which the comparative loss of motion is confined to the clavicle or to the ribs immediately over the diseased portion of lung, and to some extent also in these patients, a compensatory increase of some of the dimensions had taken place.

In conclusion, permit me to say a few words as to the stethometer used in these inquiries. The importance of the instrument for physiological purposes has, I think, been abundantly shown, even by the results which I have ventured to bring before you in this paper, and it may probably be serviceable in several other inquiries of a similar nature. We may hope that by its means the conclusions respecting the action of other respiratory muscles may be put upon a surer basis than they rest upon at present, that its use may lead to a more exact knowledge of the movements of the lower as well as of the upper ribs, and that the whole mechanism of respiration may in time be more thoroughly understood.

In medical practice also this new arm of precision may prove of great value.

The number of cases of disease hitherto examined by its aid is still too small to allow us to base upon them practical conclusions. It can scarcely be doubted, however, that important results must flow from such measurements as it affords.

Already there are indications that the prognosis of phthisis is more favorable when the ribs over the affected portions of lung begin to exercise their power of forward motion, that is, when the proportions between the several movements become more like those of health; and in emphysema and chronic bronchitis the probabilities of prolongation of life lessen directly with the diminution in the movements of the thorax, and we may probably judge of the extent to which these diseases have progressed by comparing the action of the lower with that of the upper parts of the chest wall. And we may, perhaps, learn at least one practical

lesson as to treatment from the facts which have been noted respecting the mechanism of respiration in disease.

In the first stage of phthisis it has been shown that the ribs over the irritated lung substance are deprived of one part of their motion, that, namely arising from the bending of the rib, and although this loss of movement may be partly due to the consolidation or loss of elasticity in the lung tissues; yet from the fact that it is one of the earliest symptoms of disease, and from the analogy of nature's operation in the case of other movable parts when they become inflamed, it seems probable that the want of motion is greatly due to cessation of the action of the intercostal muscles, and that it is intended to give rest to the underlying parts.

There is ample proof that independent *action* on the part of these muscles is possible, and hence there may be an independent temporary *cessation* from action. May we not then follow the guidance of nature in thus imposing rest upon the affected parts. The good effects of entire rest in bed during the acute stages of lung disease is well known.

Mr. Hilton, in his admirable work on 'Rest and Pain,' has boldly suggested the application to medical cases of the principle of Mechanical rest, which is so important in the treatment of surgical disorders. We have, also, in the facts brought forward in this paper, examples of the method adopted by nature.

It would, therefore, surely be a rational proceeding to endeavour, as much as possible, to restrict the movements of the ribs over the portions of lung affected by acute inflammation or irritation. In the cases of pleurisies and pneumonias by the application of carefully devised jackets or bandages, and in phthisis of the upper lobes by divesting the lower ribs of all ligature, and, perhaps, by the use of pads made to fit over the upper ribs and to restrain their movements.

To the consulting physician the numerical records of this instrument obtained at different stages of the various lung diseases would supply accurate data upon which to found his

opinion on the probable course of the cases brought before him, and he could refer with confidence to his notes respecting them without the vagueness which necessarily attends any mere verbal description of the results of unaided palpation.

In hospital teaching I could conceive that it would prove of distinct value, not only by enabling a clinical teacher to demonstrate the fact of alterations in the mobility of different parts of the thorax, and to point to the directions in which the motions are impaired; but it would be an additional means of inculcating those habits of exact observation and of definite measurement of morbid changes which are or ought to be the characteristic of modern medical research.

PROGRESSIVE MUSCULAR ATROPHY

ACCOMPANIED BY

MUSCULAR RIGIDITY AND CONTRACTION OF JOINTS.

EXAMINATION OF THE BRAIN AND SPINAL CORD.

BY

J. LOCKHART CLARKE, M.D., F.R.S.

COMMUNICATED BY

SIR WILLIAM GULL, BART., M.D., F.R.S.

(Received October 29th—Read November 12th, 1872.)

THE following clinical history of the case was communicated to me by Mr. Stephen Mackenzie of the London Hospital.

"Henry W—, æt. 60, was admitted into the London Hospital under the care of Dr. Ramskill. His family history was good; he was one of thirteen children, all of whom lived to a good old age, with the exception of those who died of fevers and acute diseases. He worked in a velvet manufactory, and the part of the process in which he was employed involved the use of a machine which caused a succession of knocks to his epigastrium, which often made him feel faint, and produced, he believed, continued indigestion. This employment he followed for forty years.

Up to the age of thirty he enjoyed good health, but at

this time he was seized with vertigo, for which he attended this and St. Bartholomew's Hospital and which continued, on and off, for three years. From this time he found that he was obliged to be very slow in all his movements, and that if he attempted to move his arms and legs quickly, they trembled very much.

In May, 1869, he observed that his legs gave way under him, sometimes, when he walked. Without feeling in any way giddy, he would sometimes fall down, but picked himself up and went on again. He thought the left leg was the first to be affected, and first noticed wasting in the calf of this leg. Then the right leg wasted, and four months later he noticed wasting of the left shoulder. When asked if he had pain at this time, he said he had pain in the loins, but laid great stress upon a severe dragging pain in the arms and legs, which he likened to "his veins being dragged out" or to, "strings drawing up his arms" especially increased on movement.

On admission he was unable to stand, or to turn or move himself in bed, so that he lay in one position until shifted by the nurse. He was quite incapable of feeding himself or, indeed, of moving his arms. He was able to move his fingers just a little. His forearms were kept constantly flexed on his arms, his hands on his forearms with fingers contracted, and the knees slightly drawn up.

The whole of his muscles from the head downwards were much wasted, especially those of the upper extremities, with the pectorals and deltoids. The interossei and muscles of the thumbs were not disproportionately affected.

A fibrillar tremor was seen running more or less constantly down the arms and legs. All his muscles were very rigid and this rigidity could not be overcome. When attempts were made to move his arms and legs, great pain was caused, which he always compared to "the veins being dragged out."

The respiratory movements were very feeble. He had no facial paralysis and said that he had not had any trouble with his bladder. There was no alteration of cutaneous

sensibility, but electric sensibility was greatly diminished, almost annulled.

His speech was peculiar, being indistinct and interrupted by pauses, with a slight nasal twang. He was a cheerful good tempered old man, and eat well, but slept badly.

The atrophy of muscles increased slowly at first, but rapidly at last. When he came into the hospital he was sat propped up in a chair part of the day, but about November this fatigued him so much that it was discontinued.

His respiratory movements became more and more feeble, swallowing became difficult, and at last almost impossible. The nasal character of his speech, but little noticed on admission, became more and more marked, and at last he became wholly unintelligible. Saliva ran from his mouth and, he said, the tears from his eyes. In this miserable condition he lingered for some weeks, and died on December 18th, 1871.

There are two points of great interest not noticed above. The fibrillar tremor which was so marked on admission, disappeared about the end of September, and the rigidity which was such a prominent feature of the case from the very beginning, wholly disappeared during the last week of his life."

The parts which I received for examination consisted of a slice of one of the cerebral hemispheres, the cerebellum, pons Varolii, medulla oblongata, and spinal cord.*

On examining sections of the cerebral convolutions, the white substance was found to be rather thickly interspersed with corpora amylacea varying from about twice the diameter of a blood disc to fourteen times that size. In the gray substance only a few of these bodies were present, and they were confined chiefly to the deeper layers. Many of the blood-

* On inquiring of Mr. Stephen Mackenzie whether the brain and semilunar ganglion had been examined at the autopsy, I was informed that in the brain there were no naked-eye appearances of disease beyond slight wasting of the convolutions. The brain substance was rather firmer than natural; the solar plexus and semilunar ganglion were not examined. On examination under the microscope nothing abnormal was observed in the voluntary muscles.

vessels of the white substance were enlarged, but with almost an entire absence of the granules of hæmatoidin, which I have generally found so abundant in the perivascular sheaths of the dilated vessels in general paralysis and some other cerebral diseases.

The cells of the grey substance were not altogether healthy. Some of them had lost their natural sharpness of outline, others contained rather more pigment than usual, or were somewhat granular at their surfaces.

The pons Varolii was below the average size, but presented nothing unusual in external appearance. In transverse sections, however, examined under the microscope, it was readily seen that many of the blood-vessels were much dilated. In some instances they had undergone partial disintegration at particular points, and in others they had wholly disappeared, leaving large empty and smooth-walled tubular spaces, which, according as they were cut transversely or obliquely, presented the appearance of round or oval vacuities, such as I first described in a case of general paralysis of the insane.* At nearly all parts of the sections, and particularly in the white portions, corpora amylacea were rather thickly but uniformly scattered. Moreover, the cells of the common nucleus of the abducens and facial nerves, of the motor nucleus of the trigeminus, as well as those scattered amongst the arciform fibres and plexus of the pons, had undergone more or less pigmentary degeneration.

The medulla oblongata was about one fifth below the average adult size. The hypoglossal, spinal accessory and vagus nuclei were notably smaller than usual. Like the pons, the medulla oblongata was interspersed throughout with corpora amylacea of different sizes, and the groups of cells constituting the several nuclei were more or less affected by pigmentary degeneration. Those of the gray tubercle of Rolando (caput cornu posterioris), and those especially of the restiform body suffered most. The latter under a low magnifying-power had the appearance of a chocolate-coloured mass, but under a higher power it was

* 'Journal of Mental Science,' January, 1870.

seen that while many of the cells were completely filled with brown pigment, in others the pigment granules only enveloped their nuclei, or accumulated at one side of the cell, or formed a brown ring of variable breadth at the circumference. This morbid change constitutes in many instances, as I have elsewhere shown, the first stage in the degeneration and subsequent disintegration of nerve cells.*

The diameter of the spinal cord was at least one fourth smaller than the average size in the adult. When it was sent to me without any explanation, I thought it was the spinal cord of a child about fourteen years of age; but there was nothing else abnormal in *external* appearance. Nevertheless, the grey substance from one end to the other, was severely damaged by a variety of lesions and degenerations. In the upper cervical region on a level with the second and third pairs of nerves, all the white columns were much congested; the connective tissue between their fibres was greatly hypertrophied, with proliferation of the connective tissue corpuscles which were aggregated in small groups of different sizes at the angles of junction in the network, as represented in the posterior column P, Plate I, fig. 1, on the left side. This condition was still more marked in the posterior portions of the antero-lateral columns (L) in which many of the nerve-fibres had suffered from disintegration. In the left lateral half of the grey substance a large triangular and somewhat transparent area of distintegration (D) was found in the interior of the anterior cornu, leaving only a wall of healthy and darker tissue around it. This morbid area consisted only of small remnants of partially disintegrated grey substance, irregularly connected with each other, and forming together a kind of reticular or honeycomb structure. On the right side a large area of transparent disintegration involved the whole outer half of the anterior cornu, (D) and reached as far back as the projecting group of cells, (*t, i, l,*) which I named the *tractus inter-*

* Beale's 'Archives of Medicine,' No. xiii, vol. iv, 1863, "A Case of Rapid Wasting-palsy from Disease of the Spinal Cord," p. 41, plate vi, fig. 30.

medio-lateralis, and showed to be connected with the lower rootlets of the spinal accessory nerve.* At the base of the anterior cornu and the lateral boundary of the pyriform *posterior vesicular column* (*p, v c*), was a comparatively large and fusiform hæmorrhagic clot, the outer end of which extended into the *tractus intermedio-lateralis*. Immediately behind this was a larger pyramidal clot; and still further back, at the inner angle of the *caput cornu posterioris*, and involving part of the gelatinous substance (*g*), was a smaller clot connected with a blood-vessel which extended into the posterior column.

A little lower down in the cervical region (Plate I, fig. 2), the right lateral half of the grey substance was displaced or depressed towards the corresponding side. The *tractus intermedio-lateralis* (*t, i, l*), a large portion of the cervix cornu posterioris (*d*), and the outer half of the anterior cornu were destroyed by disintegration. In the left lateral half of the grey substance, the same kind of lesion occupied nearly the corresponding parts, but in a less advanced state.

The cervical enlargement of the cord was so much atrophied that its diameter scarcely exceeded that of the portion above it. Yet the areas of disintegration were not so extensive; they were smaller and more irregular, but mostly in the corresponding localities.

In the dorsal region of the cord, (Plate I, fig. 3), both the grey and white substances were much congested, and many of the blood-vessels were dilated; but the areas of disintegration were less, and confined chiefly to the lateral border of the grey substance, involving the *tractus intermedio-lateralis*.

The diameter of the lumbar enlargement was not much below the average, nor did its external appearance indicate the existence of disease; yet the grey substance here was quite as severely damaged as in the cervical region. In (Plate I, fig. 4), on the left side, a large and increasing area of softening and distintegration is seen to extend from the outer side, *d*, of the base of the caput cornu posterioris to the

* 'Phil. Trans.,' 1859.

middle of the anterior cornu; and on the right side, from the corresponding point to the *lateral* part of the anterior cornu. In these areas partially disintegrated masses of the grey substance are more or less separated from each other by lighter spaces in which the disintegration is further advanced; so that the structure presents a somewhat mottled appearance.

In all regions of the cord the nerve cells of different parts, but particularly of the anterior grey substance, had undergone considerable degeneration and disintegration. Some were completely, others only partially, filled with dark-brown pigment granules, which in many instances enveloped and concealed their nuclei. Sometimes the pigment granules were irregularly grouped within the cells, as at *a* fig. 5; or they accumulated at one end, or at both ends of an elongated cell, as at *b*; and sometimes they formed a partial or entire ring of variable depth within the circumference of the cell, leaving a transparent portion in the centre, as at *e*. All the remaining cells were considerably reduced in size, and their processes were much shrunk, as may be seen by comparison with cells from the corresponding part of a healthy cord (Plate I, fig. 6). Many of them seemed to have been lost by gradual atrophy; but it was evident that numbers had disappeared by complete disintegration. This process of destruction could be traced through all its stages. In some places a cell could be seen actually falling into a heap of granules, as at *f*, *f*; while in others the granules into which it had been disintegrated were more or less dispersed and irregularly scattered between the still-existing but wasted cells, as at *h*, *h*.

Remarks.—The symptoms of this case are very clearly explained by the morbid changes that were found in the medulla oblongata and spinal cord. The embarrassed articulation, the nasal character of the voice, the difficulty in swallowing, and the constant escape of saliva from the mouth resemble the group of symptoms which constitutes glosso-labio-laryngeal paralysis, and are explained by the morbid

changes found in the nuclei of the facial, hypoglossal, vagus, and spinal accessory nerves. The great feebleness of the respiratory movements is accounted for by the lesions that were found in the anterior and lateral grey substance of the cervical and dorsal regions of the cord, including the *tractus intermedio lateralis*, (*t*, *i*, *l*,) which I formerly showed to be connected with lower rootlets of the spinal-accessory nerve, and with the anterior spinal roots supplying the respiratory muscles. The same progressive lesion of the anterior grey substance in the dorsal and lumbar regions of course explains the paralysis of the upper and lower extremities. According to Dr. Charcot, the contraction and stiffness of the joints in similar cases are due to sclerosis of the posterior portion of the antero-lateral columns.* There certainly was in this case, as I have already stated, decided sclerosis of that portion of the antero-lateral columns.

* 'Archives de Physiologie Normale et Pathologique,' 1869.

DESCRIPTION OF PLATE I.

Fig. 1.—Transverse section of the spinal cord between the second and third cervical nerves. A, Anterior column; L, posterior portion of lateral column; P, posterior column, showing hypertrophy of its connective tissue; *g*, the gelatinous substance; *p*, *v*, *c*, the posterior vesicular column; *t*, *i*, *l*, the *tractus intermedio-lateralis*; *d*, *d*, the anterior cornu of grey substance, in a state of softening and disintegration. Behind *d* on the right side are three comparatively large hæmorrhagic clots.

Fig. 2.—Transverse section of the grey substance of the cord a little lower down in the cervical region. The same letters indicate the corresponding parts.

Fig. 3.—Transverse section of the grey substance in the dorsal region of the cord.

Fig. 4.—Transverse section of the grey substance of the lumbar enlargement of the cord. *d*, Outer side of the base of the caput cornu posterioris. On the right side a dilated blood-vessel is seen in the middle of the softened and partially disintegrated anterior cornu.

Fig. 5.—Nerve-cells from the anterior cornu of the lumbar enlargement in variable states of degeneration and disintegration. Some are completely, others only partially, filled with brown pigment. They are all considerably shrunk, and their processes are much wasted, as may be seen on comparison with Fig. 6.

Fig. 6 represents nerve-cells from a corresponding part in a normal state, and magnified to the same extent. Some have disappeared by falling into a heap of particles as at *f*, *f*; in other places these particles have become scattered between the still-existing cells, as at *h*, *h*.

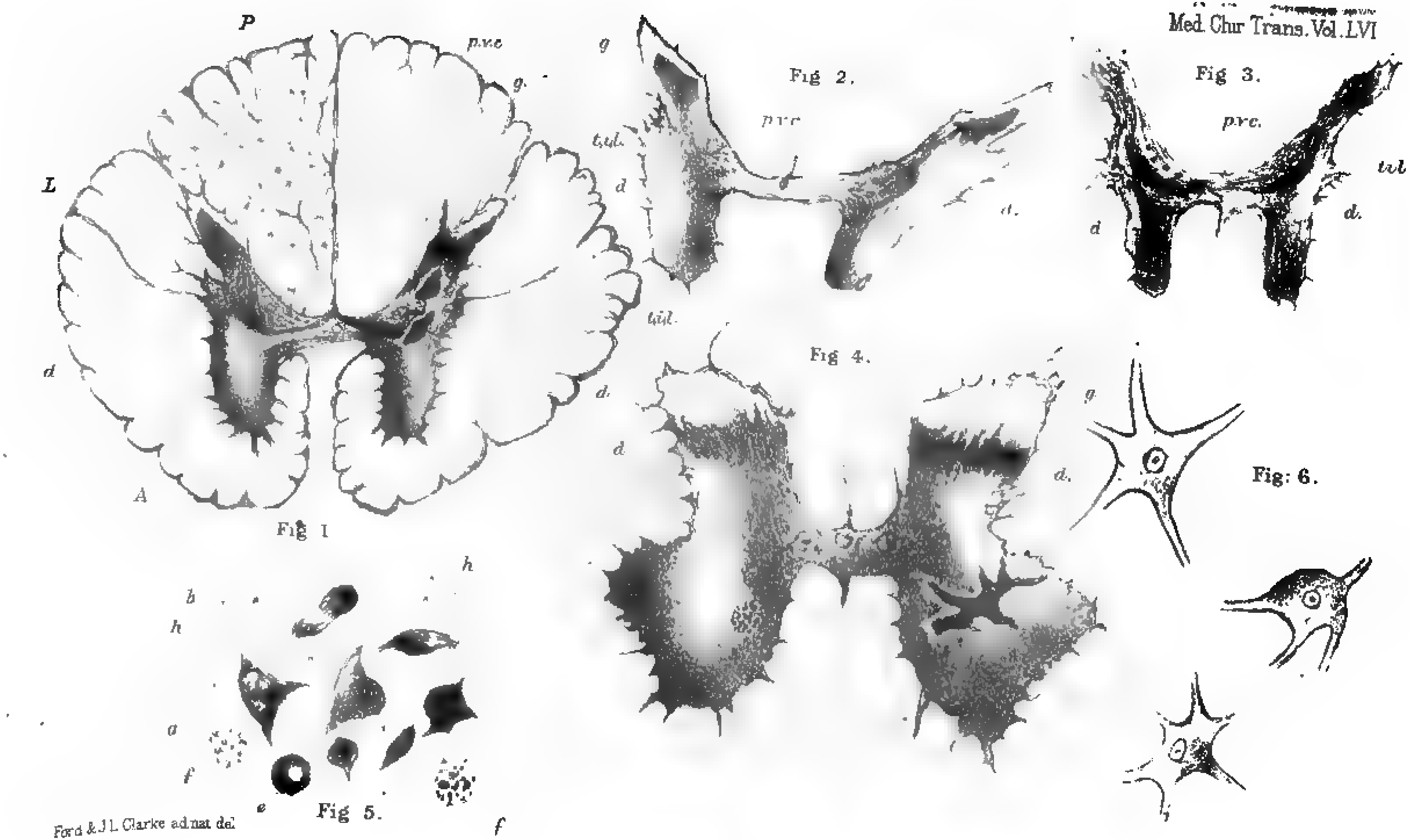


Fig 1
Fig 2.
Fig 3.
Fig 4.
Fig 5.
Fig: 6.

Ford & J. L. Clarke ad nat. del.

Mintern Bros. Chromo-lith

A FIFTH SERIES

OF

ONE HUNDRED CASES

WITH REMARKS ON THE RESULTS OF

FIVE HUNDRED CASES OF OVARIOTOMY.

BY

T. SPENCER WELLS, F.R.C.S.,

SURGEON TO THE QUEEN'S HOUSEHOLD AND TO THE SAMARITAN HOSPITAL FOR WOMEN.

Received October 8th—Read November 26th, 1872.

THE completion of five hundred cases of one capital operation by one operator offers a favorable opportunity for reviewing the progress of the operation, and our knowledge of the conditions which call for its performance, during the fourteen years which have passed since the first case in 1858 until the five hundredth this year. Looking back on this accumulated experience it may be possible to learn some useful lessons, to detect mistakes in the past which may be avoided hereafter, and to agree upon some plan of combined observation which may lay the foundation for a more complete history of ovariotomy, a more correct statistical account of its results, and a more accurate knowledge of the subsequent condition of patients who recover after it, than the records of surgery afford with regard to any other capital operation.

Particulars of all the cases from 1 to 400 have been given in successive papers which have been published in the forty-sixth, forty-eighth, fiftieth, fifty-second, and fifty-fourth volumes of the 'Transactions' of this Society. The fifth series of 100 cases is arranged in the following table :

Table of One Hundred Cases

No.	Medical attendant.	Date of operation.	Age.	Condition.	Adhesions.
		1871			
401	Mr. Yate, Godalming	Jan.	52	Married	Parietal
402	Mr. Aikin	Jan.	37	Married	Parietal
403	Hospital	Jan.	38	Married	Omental and mesenteric
404	Dr. Druitt	Jan.	63	Single	None
405	Dr. Webb	Jan.	58	Married	Parietal—Cyst suppurating
406	Dr. Sieveking	Feb.	50	Single	None—Burst cyst
407	Hospital	Feb.	25	Married	Parietal
408	Dr. Chepmall	Feb.	27	Single	Parietal
409	Dr. Webb	Feb.	21	Single	None
410	Hospital	March	32	Married	Omental
411	Mr. Weekes, Hurstpierpoint	March	25	Single	Omental
412	Hospital	March	30	Single	Parietal
413	Hospital	March	36	Single	Parietal
414	Hospital	April	43	Single	Parietal
415	Mr. Butler, Guildford	April	53	Single	Omental
416	Mr. Scrase, Lewes	April	45	Married	Parietal—Burst cyst
417	Hospital	April	38	Married	Parietal and omental
418	Hospital	April	52	Single	None
419	Dr. Ross	May	38	Married	Omental and intestinal—Pregnancy
420	Hospital	May	54	Married	Parietal and omental
421	Dr. Mayer, Berlin	May	29	Married	Parietal — Both ovaries.
422	Hospital	May	51	Single	Parietal and omental
423	Hospital	May	54	Single	Parietal
424	Dr. Greenhalgh	May	38	Married	None
425	Mr. Fouracre, Hornsey	June	19	Single	None
426	Hospital	June	29	Married	Omental
427	Dr. Schetelig, Hamburgh	June	50	Married	Parietal & mesenteric—Both ovaries.
428	Dr. Jackson, Southsea	July	50	Single	None
429	Hospital	July	29	Married	Parietal and omental
430	Hospital	July	35	Married	None
431	Dr. Ronayne, Youghal	July	30	Single	None
432	Hospital	July	68	Married	Parietal

of Completed Ovariotomy.

Treatment of pedicle.	Weight of tumour.	Length of incision.	Results.	Subsequent history or cause of death.	No.
	lbs.	inches.			
Clamp	17	5	Recovered	Health very fair in 1872	401
Clamp and ligature	15	5	Recovered	Well and pregnant in 1872	402
Clamp and ligature	6	5	Recovered	Health very fair in 1872. Asthma of long standing	403
Ligature	20	5	Recovered	Well and single in 1872	404
Clamp	19	5	Recovered	Very well in 1872	405
Clamp	21	5	Recovered	Well in 1872	406
Clamp	35	5	Recovered	Child born April, 1872	407
Clamp	16	5	Recovered	Well in 1872	408
Clamp	...	4	Recovered	Health perfect in 1872. Still single.	409
Clamp	35	5	Recovered	Very well. Small hernia near cicatrix. Girl born January, 1872—labour natural	410
Clamp	13	5	Recovered	Well in 1872.	411
Clamp	23	5	Recovered	Died April, 1872, of acute rheumatism and endo-carditis	412
Clamp	6	4	Died, 5th day ...	Septicæmia	413
Cautery	39	5	Recovered	Died in 1872 of bronchitis	414
Clamp	7	4	Died, 3rd day ...	Exhaustion	415
Ligature	34	6	Recovered	Very well in 1872	416
Clamp	24	5	Recovered	Health good in 1872	417
Ligature	7	4	Recovered	No return	418
Ligature	32	5	Recovered	Child born, Dec. 1871, 7 months after operation. Well in 1872	419
Clamp	22	5	Died, 13th day	Pleuritic effusion	420
Clamp and ligature	19	5	Recovered	Well in 1872	421
Clamp	19	4	Recovered	Well and single in 1872	422
Clamp	42	6	Died, 5th day ...	Septic peritonitis	423
Clamp	33	6	Died, 32 hours...	Exhaustion	424
Clamp	19	6	Recovered	Health very good in 1872. Still single.	425
Clamp	18	7	Died......	Went home, but died 25 days after	426
Ligature	...	9	Died..............	Peritonitis	427
Ligature	...	5	Recovered	Well in 1872	428
Clamp	30	6	Recovered	Well in 1872	429
Clamp	17	5	Recovered	Quite well in 1872	430
Clamp	22	4	Recovered	Well in 1872	431
Clamp	25	6	Recovered	Well in 1872	432

No.	Medical attendant.	Date of operation.	Age.	Condition.	Adhesions.
		1871			
433	Hospital	July	50	Married	None—Both ovaries
434	Professor Winkel, Rostock	Aug.	37	Married	None
435	Dr. Bell	Aug.	42	Married	Parietal
436	Mr. Barlow	Aug.	41	Married	Omental & mesenteric
437	Dr. Boddaert, Ghent	Aug.	52	Married	Parietal and pelvic...
438	Mr. Ticehurst, Hastings	Aug.	22	Single	None
439	*Hospital*	Aug.	32	Single	Parietal and omental —Burst cyst
440	*Hospital*	Aug.	23	Single	None
441	*Hospital*	Oct.	41	Married	None—Both ovaries
442	Mr. Baker, Birmingham	Oct.	32	Single	None—Burst cyst ...
443	*Hospital*	Oct.	42	Single	None
444	Dr. Farre	Oct.	50	Married	None
445	Dr. Budd, Clifton	Nov.	30	Single	None
446	Dr. Pirrie, Belfast	Nov.	40	Married	None
447	Mr. Marriott, Leicester	Nov.	42	Married	None
448	*Hospital*	Nov.	27	Single	None
449	Dr. Lyon, Clifton	Nov.	29	Single	None
450	Mr. Roughton, Kettering	Nov.	42	Married	None
451	Mr. Riggall	Nov.	56	Married	Parietal—Cyst suppurating
452	Hospital	Dec.	27	Married	None
453	*Hospital*	Dec.	34	Single	None
454	Hospital	Dec.	40	Married	Parietal
455	Hospital	Dec.	21	Single	None
456	Hospital	Dec.	28	Single	None
457	Sir J. Alderson	Dec.	60	Married	Intestinal and mesenteric
		1872			
458	Dr. Turner, Minchinhampton	Jan.	27	Married	Parietal
459	Hospital	Jan.	17	Single	Parietal and omental
460	Mr. Bell, Rochester	Jan.	60	Single	Parietal & intestinal
461	Mr. T. H. Hill	Jan.	55	Married	Parietal and pelvic...
462	Dr. Smith	Jan.	53	Married	Parietal, omental, and intestinal
463	Mr. Turner, Bermondsey	Jan.	46	Married	None—Burst cyst ...
464	Dr. Stewart, Whitby	Jan.	48	Married	Omental and pelvic
465	Hospital	Feb.	22	Married	None
466	Mr. Pollard, Torquay	Feb.	46	Married	Parietal, omental, and intestinal
467	Dr. Powne, Swindon	Feb.	57	Married	Parietal
468	Hospital	Feb.	23	Married	None—Burst cyst ...
469	Hospital	Feb.	41	Single	None
470	Hospital	Feb.	44	Single	None
471	*Hospital*	Feb.	48	Married	Parietal and omental
472	Hospital	Feb.	44	Married	None
473	*Hospital*	March	51	Single	Parietal and omental
474	*Hospital*	March	40	Married	Omental—Burst cyst
475	Hospital	March	32	Single	Omental

Treatment of pedicle.	Weight of tumour.	Length of incision.	Results.	Subsequent history or cause of death.	No.
	lbs.	inches.			
Clamp and ligature	21	5	Recovered	Died Dec. 1871 of malignant disease	433
Clamp	12	5	Recovered	Well in 1872	434
Clamp	...	5	Recovered	Very well in 1872	435
Ligature	15	5	Recovered	Well in 1872	436
Clamp	33	6	Recovered	Well in 1872	437
Clamp	8	4	Recovered	Well in 1872	438
Ligature	...	6	Died, 5 hours ...	Collapse	439
Clamp	13	4	Recovered	Health very good in 1872. Still single	440
Ligature	37	6	Died, 3rd day ...	Pulmonary embolism	441
Clamp	11	5	Recovered	Well in 1872	442
Clamp	23	4	Recovered	Well in 1872	443
Clamp	28	4	Died, 7th day ...	Septicæmia	444
Clamp	8	5	Recovered	Well in 1872	445
Ligature	24	5	Recovered	Well in 1872	446
Clamp	23	4	Recovered	Well in 1872	447
Clamp	8	4	Died, 5th day ...	Septicæmia	448
Ligature	18	4	Died, 5th day ...	Hyperpyrexia and pericarditis	449
Clamp	15	5	Died, 23 hours	Exhaustion	450
Clamp	49	8	Died, 26 hours	Septicæmia	451
Clamp	35	6	Recovered	Remains well	452
Clamp	11	5	Died, 4th day ...	Septicæmia	453
Clamp	51	6	Recovered	Remains well	454
Clamp	16	5	Recovered	Remains well	455
Pin and ligature......	10	5	Recovered	Remains well	456
Clamp	15	6	Recovered	Remains well	457
Clamp	22	6	Recovered	Remains well	458
Clamp	16	5	Recovered	Remains well	459
Clamp	33	6	Recovered	Died in March	460
Clamp	10	4	Recovered	Remains well	461
Clamp	18	5	Recovered	Remains well	462
Clamp	...	4	Recovered	Remains well	463
Clamp	41	6	Died, 3rd day ...	Exhaustion	464
Clamp	24	4	Recovered	Remains well	465
Clamp	15	4	Recovered	Remains well	466
Clamp	14	5	Recovered	Remains well	467
Clamp	36	5	Recovered	Remains well	468
Clamp	15	4	Recovered	Remains well	469
Clamp	16	4	Recovered	Remains well	470
Clamp	33	5	Recovered	Remains well	471
Clamp	28	5	Recovered	Remains well	472
Clamp	19	5	Died, 4th day ...	Peritonitis	473
Ligature	16	5	Died, 3rd day ...	Peritonitis	474
Ligature	30	5	Died, 7th day ...	Peritonitis	475

No.	Medical attendant.	Date of operation	Age.	Condition.	Adhesions.
		1872			
476	Hospital	March	29	Married	Omental—Pregnancy
477	Hospital	March	50	Married	None—Burst cyst
478	Hospital	April	26	Single	Omental
479	Hospital	April	31	Married	Parietal and omental
480	Hospital	April	23	Single	Parietal
481	Professor Bardeleben	April	24	Single	None
482	Mr. Lys, Blandford	April	27	Married	Parietal
483	Hospital	April	48	Married	Parietal
484	*Hospital*	April	60	Married	Parietal and omental
485	Dr. Day	April	43	Married	None
486	Mr. Earle, Brentwood	April	48	Married	None
487	Hospital	April	57	Married	Parietal
488	Sir W. Gull, Bart.	May	53	Single	None
489	Sir W. Gull, Bart.	May	29	Single	Omental
490	Mr. Moreton, Tarvin	May	51	Married	Parietal
491	Hospital	May	42	Single	None
492	Hospital	May	53	Married	Parietal
493	Mr. Mason	June	39	Married	Parietal
494	Hospital	June	36	Married	Both ovaries.—Burst cyst
495	Dr. Hickson, Scarboro'	June	22	Single	None
496	Hospital	June	46	Married	Parietal and omental
497	Mr. Bracey, Birmingham	June	48	Married	Parietal and omental
498	Mr. Whipple, Plymouth	June	21	Single	None
499	Hospital	June	37	Married	Parietal and omental
500	Hospital	June	54	Married	Parietal and omental

Treatment of pedicle.	Weight of tumour	Length of incision.	Results.	Subsequent history or cause of death.	No.
	lbs.	inches.			
Ligature	10	5	Recovered	Child (girl) born at 6th month; lived 21 hours; mother quite well.	476
Clamp and ligature	17	7	Died, 4th day ...	Pneumonia	477
Clamp	20	6	Recovered	Remains well	478
Clamp	24	4	Recovered	Died, July, 1872 — obstructed intestine	479
Clamp and ligature	25	7	Recovered	Remains well	480
Clamp and ligature	12	6	Recovered	Remains well	481
Clamp	27	5	Recovered	Remains well	482
Clamp	...	5	Recovered	Remains well	483
Clamp	26	7	Recovered	Remains well	484
Clamp	8	5	Recovered	Remains well	485
Clamp	14	4	Recovered	Remains well	486
Clamp	...	5	Recovered	Remains well	487
Clamp	22	4	Recovered	Remains well	488
Clamp	26	5	Recovered	Remains well	489
Clamp	28	4	Recovered	Remains well	490
Clamp	...	6	Recovered	Remains well	491
Clamp	...	5	Recovered	Remains well	492
Clamp	34	5	Recovered	Remains well	493
Clamp	21	4	Recovered	Remains well	494
Pin and ligature......	18	5	Recovered	Remains well	495
Ligature	25	6	Recovered	Remains well	496
Clamp	26	5	Recovered	Remains well	497
Clamp	6	4	Recovered	Remains well	498
Clamp	16	5	Recovered	Remains well	499
Ligature	24	5	Recovered	Returned to Suffolk. Died a month after with cerebral symptoms	500

I do not propose now to make any remarks upon this series of cases alone, but to group the 500 cases together, and to consider what may be learned from a review of the whole.

Following the order of the successive columns of the table, we may first inquire how the results of ovariotomy are influenced by the social condition of the patient, so far as this can be ascertained by the fact of the operation having been performed in hospital or in private practice. It should be stated that all the cases marked "Hospital" in the first column, with the exception of ten in which the word *Hospital* is printed in italics, have been in the Samaritan Hospital, which in many respects resembles a private house, having until this year scarcely ever had more than eighteen beds, and since the beginning of this year only about twenty-four beds occupied. The ten exceptional cases were in a nursing institution in Duke Street, Portland Place, a house where the separation of patients is rather more complete than in the Samaritan Hospital, where the nursing is excellent, but where the results were not nearly so good; the ten cases showing only four recoveries to six deaths. The social condition of some of the patients has been higher than that of the women in the Samaritan Hospital; but in all other respects they may be classed more correctly among the hospital patients than among those who had had the advantage of a separate room in a house free from other patients, and the undivided attention of a nurse. Including these ten cases there have been 240 in hospital, and 260 in private practice. The mortality has been 26·66 in hospital and 24·23 in private practice, a difference of only 2·43 per cent. in favour of private patients upon the whole number. In the first 100 there was a balance of 10 per cent., and in the third 100 of 4 per cent. in favour of hospital cases. In the fourth 100 the mortality in hospital was more than double that in private practice, the proportions being 31·84 and 14·28. In this fifth series, the general mortality being lower than in any of the preceding series of 100 cases, the hospital mortality was 21·05, while the private was 16·32. On endeavouring to

ascertain the reason for this varying proportion of mortality in hospital, it has become manifest that periods of high and low mortality have corresponded with varying sanitary conditions in the hospital, or with the presence of infecting cases in some ward. After emptying the house for a month or longer, thoroughly cleansing, painting and lime-whiting the wards, a period of almost uninterrupted success has followed. Then what some call "a run of bad luck" set in, attributable, I believe, to crowding, to some neglect in purifying bedding, or to contagion or infection. Another thorough cleansing was again followed by better results, and in the six months from December, 1871, after complete repairs, till July, 1872, of 24 cases in the Samaritan Hospital only 2 died and 22 recovered. During the same period in private practice there were 21 successful cases without one death; and I have reason to fear that in some of the earlier private cases a better result might have been obtained if more attention had been paid to the sanitary condition of the house, its drains, sinks, closets, and water supply, while I and all those about the patients had been perfectly free from any infecting influence. My conviction is that the surgeon who hopes to obtain better results than have hitherto been obtained must place his patient, whether in hospital or private practice, as nearly as he can in the position of a person in a private house in a healthy situation, in a room where ventilation is sufficient and continual but not excessive, and the temperature is under proper regulation, all unnecessary furniture (such as woollen carpets and curtains) removed, all bedding and clothing being perfectly clean and free from any taint of morbid poison, and the patient having the undivided attention of one trustworthy nurse for several days after operation.

In the second column of the table the date of the operation is given, thus enabling us to examine the *influence of season* upon the result of operations, and it seems that the mortality is nearly equally distributed over the whole year. It appears probable, therefore, that if atmospheric and climatic conditions do affect the mortality, these conditions, whatever they may be, are not more prevalent in

any one month, or in any season of the year, than in others.

The influence of *age*, so far as can be learned from a study of the facts in the succeeding column, appears to be remarkable. Thus patients below the age of 20 and above that of 60 have almost all recovered. Of twelve under 20 every one recovered, and of 8 above 60, seven recovered, or of those 20 very young and very old patients, only one died. Between 20 and 25, and between 40 and 45, the mortality was about the same, namely, 17 and 16 per cent. The highest, 33 per cent., was at the age of 50 to 55.

The *conjugal condition* of the patient appears to have but little influence upon the mortality. The number of married patients was 279, of unmarried 221. In the married the mortality was 26·84; in the unmarried 23·52 per cent., showing a difference of 3·32 in favour of unmarried over married women at all ages.

Influence of adhesions.—The general conclusion at which I have arrived is that while adhesions to the abdominal walls or to the omentum have but little influence upon the mortality after ovariotomy, adhesions within the pelvis or around its brim which endanger the iliac vessels and the ureters, and attachments to the bladder or rectum, which are separated with difficulty and may lead to delay in securing bleeding vessels, are likely to considerably increase mortality. Thus, the diagnosis of adhesions within the pelvis before operation becomes of considerable importance, while the diagnosis of adhesions to the abdominal wall may be practically disregarded. Statistically, we may say that of the 500 cases, in 296 women there were either no adhesions, or they were so slight as to be of no importance. In these cases the mortality was 19·93 per cent. In 204 cases, where the adhesions were very extensive or were within the pelvis, the mortality was 33·33 per cent., or about 13 per cent. higher than in the cases where adhesions were not met with or were but slight.

The *size* of a tumour, its *solidity*, and the weight of its contents must be considered with the length of the incision

required for removal. For, when the contents of either single or multilocular cysts can be evacuated after tapping or breaking up the septa, very large tumours may be removed through a small opening; whereas a long incision may be necessary for the removal of a solid tumour of moderate size. Looking for the results of incisions of different lengths, it appears that the smallest mortality has been in cases where the incision was about five inches long. This is long enough to admit one of the operator's hands easily and remove an empty cyst or a broken-up tumour without force. Of 203 such cases the mortality was 19·7 per cent. In 140 cases of a four-inch incision the mortality was 24·28 per cent. In 17 cases, where the incision was only three inches long, the mortality was 23·53 per cent. If we compare these 360 cases where the incision did not exceed five inches, the mortality being 21·66 per cent., with the 140 cases where the incision was six inches and upwards, the mortality having been 35 per cent., it appears that the mortality has increased with the length of the incision after five inches. Incisions of six inches show a mortality of 31·25, seven inches 34·48, eight inches 35·0, nine inches, 50·0; and of three cases where the incision was ten and twenty inches, all died—not that the greater length of incision of itself adds much to the danger, but that it is an index of the size and solidity of the tumour and of the difficulty attending its separation and removal.

The *treatment of the pedicle* is far too important a question to discuss on this occasion, but it may serve as a guide to future observers if the results of different modes of treatment in 500 cases are shortly stated. In about two thirds of the cases, or 349, the pedicle has been secured by a clamp outside the abdominal wall; of these cases 280 recovered and 69 died, a mortality of 19·77 per cent. This represents the mortality of the *extra*-peritoneal method of treating the pedicle when this method is completely carried out. The strangulated portion of the pedicle is held up by the clamp outside the abdominal wall, and does not sink inwards between the divided edges of the skin. If an

attempt be made to carry out the extra-peritoneal method in any other way, either by securing part of the pedicle with a clamp, and the remainder by ligature, or by transfixing the pedicle with a pin and securing it with a ligature behind the pins which is either passed through the skin or fastened outside like a clamp, or by sewing the pedicle to the abdominal wall; there is far less complete security against sinking inwards of the strangulated dying portion of the pedicle between the lips of the wound or even into the peritoneal cavity. Such substitutes for the clamp have been tried in 49 cases, with a result of 33 recoveries and 16 deaths, a mortality of 32·65 per cent., a difference of about 13 per cent. in favour of the complete over the imperfect method of carrying out the extra-peritoneal principle. But even these imperfect attempts to practise the extra-peritoneal method have given far better results than the old method of tying the pedicle, allowing it to sink into the peritoneal cavity with the ligature, and keeping the lower part of the wound open by the ends of the ligature until it separates and comes away. Of 14 cases so treated, 8 died and 6 recovered,—a mortality of 57·14 per cent.

The *intra*-peritoneal method has been practised in 88 cases with a result of 54 recoveries and 84 deaths, or a mortality of 38·63 per cent., just double that of the *extra*-peritoneal method when carried out by the clamp. In 57 cases the tied pedicle was returned into the peritoneal cavity with the ligature securing it, the ends being cut off short, and the wound closed; 29 of these patients recovered and 28 died—a mortality of 49·12 per cent. The *écraseur* was only used in one case, and the patient recovered. The cautery alone was used in 16 cases, with a result of 14 recoveries and 2 deaths. In 14 other cases, where the cautery was used, ligatures also became necessary to one or more bleeding vessels not closed by the cautery. The results were 10 recoveries and 4 deaths; or, grouping together all the cases where the cautery was used, we have 24 recoveries and 6 deaths, a mortality of 20 per cent.—within a

fraction of the 19·82 per cent. of this mortality in the clamp cases.

Included among the 500 cases of ovariotomy are 25 cases where both ovaries were removed at the one operation. If these 25 cases were deducted from the 500 the number of cases of removal of one ovary would be 475, and the deaths 115, a mortality of 24·44 per cent.; and as 11 died of the 25 cases of double ovariotomy, the mortality has been 44 per cent., or nearly double that of the single cases. In double as in single ovariotomy the chief point of importance is the mode of dealing with the pedicles, and the results of my experience are in favour of the *extra*-peritoneal method of treating both pedicles whenever it is possible.

In four cases not included in the list of 500, ovariotomy has been performed twice on the same patient. Recovering after removal of one ovary, the patient some months or years afterwards became the subject of disease in the other ovary, and underwent a second time the operation of ovariotomy. Two recovered and two died after this second operation.

In all the previous papers published in the 'Transactions' of this Society, I have given, in addition to the table of cases of completed ovariotomy, another table of cases where the operation was commenced, but could not be completed, and of cases where an exploratory incision was made to complete a doubtful diagnosis. The following table gives the particulars of seven cases of this kind which were observed during the progress of the fifth series of 100 cases:

Cases in which Ovariotomy was commenced, but not completed.

No.	Medical attendant.	Date.	Age.	Condition.	History, &c.	Results.
1	Dr. Wane	1871. Aug.	63	Married	Ascitic fluid and contents of two cysts removed; papilliform masses, involving uterus, both ovaries, bladder, and rectum, undisturbed.	Died, 15 days after.
2	Mr. Coe, Bristol ...	Nov.	42	Widow	Multilocular cyst emptied and partly removed; the remainder (adherent to the rectum, uterus, and pelvis generally) stitched to the opening in the abdominal wall, which was partly left open for drainage.	Died, 10th day.
3	Dr. Barclay, Leicester.	Nov.	43	Married	A burst cyst, and ovarian fluid free in peritoneal cavity partly removed; the lower segment of the cyst, generally adherent behind the uterus, stitched to the lower edges of the wound and drained.	Died, 3rd day.
4	Dr. Millington, Wolverhampton.	1872 Jan.	55	Married	Ascitic fluid removed; a cyst emptied. Nothing more done. Nodules of cancer covering the whole peritoneum.	Recovered from operation, but died of cancer of rectum 10 weeks after.
5	Hospital	April	27	Single	Peritoneal fluid removed; bunches of thin grape-like cysts drawn out. Part of a large cyst firmly adherent in the pelvis not separated, but secured outside by a large clamp.	Died, 11 days after.
6	Hospital	Jan.	15	Single	Renal cyst emptied and drained.	Died, 4th day.
7	Dr. Sutton, Dover.	June	42	Married	Ovarian cyst tapped and emptied, but not separated from a large fibroid uterus, to which it was universally adherent. Wound closed.	Recovered from operation.—Returned to Dover. Afterwards treated by drainage.

With the whole series of 500 cases of completed ovariotomy there have been 52 cases of exploratory incisions or of incompleted operations. Of these patients 33 recovered from the operation, or were relieved by it. In 19, the patients died at various periods from one to fifteen days afterwards. In some cases about as much was gained as by ordinary tapping. Five patients recovered completely; 3 of them, after opening the cyst, fixing a catheter or drainage tube in it, and closing the wound around it, perfect recovery following suppuration and drainage of the cyst.

The subsequent history of the patients who recovered after removal of one ovary shows that they may menstruate regularly, and may bear children of both sexes, or twins; and that after removal of both ovaries they do not become excessively fat, nor lose their feminine appearance or sexual instinct. Of the 373 women who recovered, I obtained particulars this year of the condition of all but 23; and I ascertained that 36 patients who were unmarried at the time of the operation have married since. Of these 15 have had one child, 6 two, 3 three, and 3 four children. Two have had twins. Of 259 who were married when the operation was performed, many being beyond the age of childbearing, 23 have had one or more children since; 112 women were in good health this summer, many of them saying they were stronger than they had been for several years before. A few complain of some trifling ailment, and 36 have died at various periods, from a few weeks to eight years, after recovery from the operation. In 17 of these cases the cause of death was more or less directly connected, and in 19 not at all connected with ovarian disease or the operation. These cases can only be regarded as rare exceptions to the general rule that a woman who recovers after ovariotomy is restored to perfect health, and is enabled to fulfil all the duties of wife and mother.

In a work recently published in America, Dr. Peaslee, of New York, says, "It may be shown that in the United States and Great Britain alone ovariotomy has, within the last thirty years, directly contributed more than 30,000 years

of active life to women, all of which would have been lost had ovariotomy never been performed." Applying the calculations by which he arrives at this result to my own 500 cases, taking the average age of all the patients as thirty-eight years, and the average expectation of life for a healthy woman of that age in this country as twenty-nine years, we find the probable aggregate duration of life of the 373 survivors amounts to 10,817 years. Admitting that the probable average duration of life in women who have an ovarian tumour of such a size as to raise the question of removal to be four years—a very liberal estimate—the 127 who died may have lost 508 years, and the 373 who recovered might have passed 1492 years of miserable endurance, whereas they have secured by the operation the probability of the gross amount of 10,817 years of average healthy life.

CASE

OF

SUBCLAVIAN ANEURISM

IN WHICH

TEMPORARY COMPRESSION OF THE INNOMINATE WAS TRIED, FOLLOWED BY LIGATURE OF THAT VESSEL.

BY

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Received October 17th—Read November 26th, 1872.

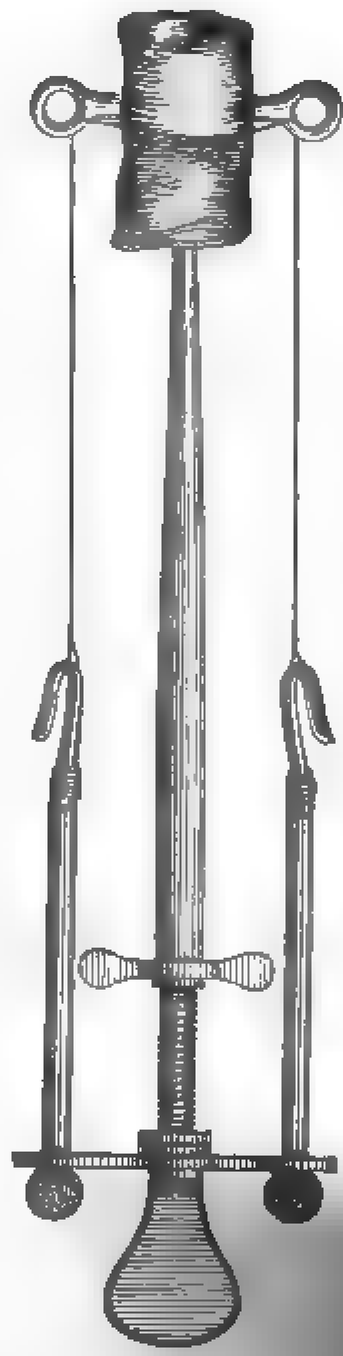
JAMES J—, æt. 40, was admitted under my care into the Liverpool Royal Infirmary on the 15th of April, 1868, suffering from an aneurism of the third part of the right subclavian artery. He was a vigorous, muscular, healthy looking man, occupied as a dock porter. He described his condition as arising from a strain while lifting a bale of cotton three weeks previously, immediately after which he felt ill-defined pains and uneasiness at the root of the neck with stiffness of the right arm, and in a few days a pulsating swelling above the collar-bone appeared. The tumour was well marked, about the size of a hen's egg, its inner margin reaching to the outer edge of the sterno-mastoid. It had an aneurismal thrill, pulsated vigorously, and admitted of being partially emptied by compression, but no distinct bruit could be detected. In short, no doubt could be entertained re-

garding the character of the tumour, and as it was increasing rapidly, while in all other respects the man appeared to be thoroughly sound, it seemed desirable to attempt some operation for his relief.

The various plans tried and suggested by surgical authorities were anxiously considered, but all appeared to be equally hopeless. Ligature on the distal side of the aneurism offered no hope of success on account of the large branches arising immediately below, which would still continue to be fed even after the trunk was tied. Ligature in the first part of the subclavian has never been attended with success, and the second portion (that between the scaleni) was in this case probably so closely involved in the sac of the aneurism that deligation there could not be attempted. On the other hand, ligature of the innominate afforded but little better hope, for of the thirteen cases collated by Mr. Erichsen, twelve proved fatal, and in the solitary instance of recovery, although the carotid was tied at the same time as the innominate, hæmorrhage occurred on the fifteenth, thirty-third day, and fifty-first days, and then, as a last resource, on the fifty-fourth day the vertebral artery was also tied, after which no more bleeding took place, and the patient, we may almost say "miraculously," recovered.

Ligature, then, as ordinarily practised, appeared hopeless; compression was obviously impossible, and in galvano-puncture I had no better hope. The success attending the complete, temporary arrest of the current of blood through an aneurism, proved in very many cases, and more especially in the notable instance of cure of a large abdominal aneurism recorded by Dr. Murray, of Newcastle-upon-Tyne, who, after effectually compressing the abdominal aorta for a few hours, found such complete coagulation in the sac had occurred as to lead to its permanent obliteration, led me to hope that if any means could be contrived by which the current of blood, flowing through the aneurism, could be temporarily yet completely arrested for a few hours in the present case, a similar result might reasonably be hoped for. The difficulty to be encountered was how to apply temporary compression

effectually, and, as the anatomical position of the vessel rendered any other means impossible, I proposed a modification of the plan adopted with the acupressure needle. Although I have long ceased to employ acupressure, there is no doubt about the fact (which I have by dissection corroborated for myself), that if a needle be passed under an artery at some distance above its cut extremity, it does not necessarily leave any trace of its presence at this point, although it has effectually and permanently controlled all hæmorrhage. I hoped, then, that by passing a wire under the artery, and making temporary compression with a metallic bar upon it, I should gain the end sought without injury to the vessel. Mr. Porter, of Dublin, recorded in the 'Dublin Quarterly Journal,' for November, 1867, a case in which he compressed the innominate by an instrument not unlike a miniature lithotrite, the blades of which, after including the artery, were screwed down so as completely to compress the vessel. Fatal hæmorrhage resulted owing to a slough having formed from the pressure. I hoped that by employing elastic instead of unyielding pressure a better result might be obtained, and accordingly the accompanying cut, which shows the instrument, was devised. The screw arrangement in



the shaft of the instrument enabled me to modify the amount of pressure at any time without disturbing the artery, and I trusted to being able to control the circulation by the gentle and yielding pressure afforded by the minute india-rubber "accumulators," without inflicting serious injury

upon the arterial coats. The chief difficulty that presented itself was the finding of a wire sufficiently tough, and yet so pliable that, when drawn home through the rings at the extremities of the transverse bar intended to rest upon the artery, it would bend easily and compress equally every part of the artery. Gold, silver, platinum, and aluminium were all tried, but not found to answer. None of them admitted of being drawn sufficiently tightly to occlude the calibre of the vessel without injury to its coats. But lead wire appeared to answer in every respect—very pliable, comparatively soft, yet tough, I resolved to place it under the innominate, and then apply elastic compression.

During the period that elapsed while making the above trials the patient was kept very quiet with his arm supported in a sling, but the tumour obviously increased, and a bruit now became audible.

Operation.—On the 5th of May, I made an incision parallel with the anterior edge of the sterno-mastoid muscle down to the sterno-clavicular articulation, and another from this point outwards along the clavicle. The flap thus formed was raised and reflected, and the sternal attachment of the sterno-mastoid was divided close to the bone. Next, the sterno-hyoid and sterno-thyroid muscles were cut across aud the lower portion of the common carotid and internal jugular became visible. Some little trouble was experienced with cross venous branches running between the anterior and external jugulars, but they were either held aside or cut across and tied. The sheath of the common carotid was there followed down to its junction with the subclavian, and innominate exposed. Its sheath was opened and an aneurism needle passed under it armed with a thread by which the lead wire was next pulled through. Then the ends of the wire were passed through the apertures in the transverse bar, and made fast to the "accumulators." The bar was pushed down upon the artery, and the wire tightened by turning the screw in the shaft of the clamp till pulsation in the aneurism and radial artery ceased. Then the wound was closed by sutures except where the instrument projected.

The operation was accomplished with remarkable facility, and almost without the loss of blood. It was interesting to observe the movements of the clamp caused by the impulse of blood on the immense vessel which was thus being controlled. No sickness or pain occurred, the patient enjoyed his food the same evening, and the pulse remained as before the operation, about 60.

May 6th.—Patient slept frequently for a short time during the night, but complained of slight pain at the epigastrium. He had some toast and beef tea. To-day he looks well and cheerful, and his pulse is quiet. A shot bag was placed over the anenrism.

7th.—During the night the patient complained of pain in the head, and also in the epigastrium. About 10 a.m. pulsation was observed in the aneurism, and Mr. Puzey, the house surgeon, proceeded to tighten the wire by turning the screw in the clamps, but, finding this did not control the pulsation, he looked into the wound, and then observed that the lead wire had broken on one side at the point where it passed through the aperture in the transverse bar. The constant sawing movement, caused by the impulse of the artery had evidently worn it through. At 1 p.m. a consultation was held, and it was decided not to replace the clamp, as the leaden wire was obviously insufficient, and no other form appeared to possess the requisite suppleness. The wound was completely opened and found to be in a very healthy state. The artery, at the spot where the pressure had been, looked a little discoloured, but seemed healthy. A double silk ligature was passed beneath the vessel at the same part where the wire had been, and was tied firmly at two points very slightly apart from each other. The two threads were employed in order to tie above and below the points where the wire had been, as it was feared that the vessel might not be in a favorable condition at this part. This second operation was accomplished easily without any loss of blood, and, after it, pulsation in the tumour and in the radial artery ceased.

8th.—Passed a good night; pulse 70. Tumour decidedly

smaller. Patient looks well and is cheerful. It was noticed that the right pupil was slightly dilated, and the wound looked a little red at the edges.

9th.—Patient looks well and cheerful. Passed a good night. The tongue is clean, and pulse 76. The wound discharged a thin, dirty matter.

10th.—All going on well. There is less redness round the wound, which, however, still discharges the same kind of matter.

11th.—Patient slept irregularly. Pulse 88. Complains of frontal headache. Radial pulse perceptible on the affected side. Wound more inflamed.

12th.—Patient slept well. Pulse 96. Tongue clean and appetite good. He still complains of the frontal pain and of numbness in the right arm. The sutures having given way were removed, and the edges of the wound brought together with adhesive plaster.

13th.—Slept well. Pulse 86. Feels comfortable. It was noticed that the carotid pulse was much more visible than before. At 7.45 p.m., during a fit of coughing some hæmorrhage took place from the wound, but was easily controlled by pressure.

14th.—At 8.45 a.m. there was another and more severe attack of hæmorrhage lasting a quarter of an hour. At 12.50 there was bleeding again lasting for eleven minutes, and at 7.45 p.m. a fearful gush of blood occurred, the patient became convulsed and died in about an hour. After the bleeding in the morning, the wound was opened and cleansed and its cavity filled up with loose shot, after which a bag containing shot was placed over it.

Post-mortem examination.—The heart, aorta, great veins and arteries, trachea and all the structures on the right side of the neck, including the aneurism were removed *en masse*, and carefully dissected out. The heart was found to be quite healthy, but the aorta exhibited considerable signs of atheromatous degeneration in its early stage. The innominate, from its origin from the aorta to the point of ligature, was filled with a firm closely-fitting plug of fibrin—was, in fact,

most satisfactorily occluded. Above the ligature to its bifurcation the vessel was quite empty, while the common carotid and the subclavian as far as the aneurism were also quite empty and did not contain a trace of clot. The aneurism had obviously diminished a good deal in size and was quite filled with firm laminated clot. Indeed it may be said to have been in fair progress of cure. The rest of the subclavian beyond the aneurism and first part of the axillary artery, were nearly, but not quite filled up with clot. So that from the aorta to the ligature and from the aneurism onwards clotting had occurred, while between the ligature and aneurism this had not taken place. The two ligatures were found close together and still *in sitû*, and the hæmorrhage was seen to have proceeded from the distal side of the upper of the two ligatures, which had partially cut through the vessel. The wound, although not in a healthy state, did not present an unhealthy aprearance. All the other viscera were quite normal.

The actual condition of the parts is shown in the drawing (see Plate II) from the preparation exhibited at the meeting.

In presenting this case to the Society I must claim its indulgence. I cannot feel satisfied that even the process of reasoning which led to my plan of operation was free from error. It was my intention to have removed the clamp and wire at the end of forty-eight hours, when I hoped that a fibrinous plug sufficiently firm to secure the subsequent obliteration of the aneurism would have formed, and I trusted to do this without injury to the coats of the innominate, but the return of vigorous pulsation in the tumour after forty-five hours of compression too clearly indicated that my expectations were miscalculated. Although I do not think that any injury had, up to this time, been inflicted on the innominate coats, it was evidently endangering their vitality to continue the compression any longer, and I was thus compelled, as it were, to resort to the ordinary process of deligation. Possibly, had I then desisted from further interference, the artery might have contracted and closed from adhesive inflammation.

But while deploring the result which I have thus thought it my duty to detail and while resolved not to attempt again a like procedure, I am by no means satisfied that deligation of the innominate will not become a justifiable and proper operation in cases of aneurism of the subclavian. At the time when the case narrated occurred, antiseptic surgery was yet in its infancy, and the marvellous results to be obtained by the catgut ligature properly prepared and the subsequent antiseptic treatment of the wound were unknown. From the experience and confidence I have obtained in this method of tying the iliac, carotid, and femoral arteries, I should feel justified in attempting again deligation of the innominate in a similarly otherwise hopeless case.

DESCRIPTION OF PLATE II.

A.—Aneurism laid open, showing laminated clot completely filling interior.

B.—Subclavian artery between innominate and aneurism quite empty.

C, D.—Lower portion of carotid and upper part of innominate laid open, showing absence of clot.

E.—Firm fibrinous clot filling up innominate from its origin to the point of ligature.

F, G.—Subclavian beyond aneurism and upper part of axillary artery nearly filled with clot.

G. H. Ford. Mintern Bros. Chromo lith.

THE PATHOLOGY

OF

CHRONIC BRIGHT'S DISEASE WITH CONTRACTED KIDNEY,

WITH ESPECIAL REFERENCE TO THE

THEORY OF "ARTERIO-CAPILLARY FIBROSIS."

BY

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In the fifty-first volume of the 'Medico-Chirurgical Transactions' I published a paper in which I described and figured what I look upon as a genuine hypertrophy of the muscular walls of the minute arteries from various tissues in cases of chronic Bright's disease. I there stated that I had observed a constant relation between hypertrophy of the arterial walls and hypertrophy of the left ventricle of the heart. And I gave what I believe to be the true physiological interpretation of the phenomena. I have looked upon this discovery of hypertrophy of the arterial walls as the most interesting result of a long series of observations extending over a period of rather more than a quarter of a century, and I supposed that my doctrine relating to this particular subject had met with very general assent. In this belief, however, I now find that

I was mistaken. The recently issued volume of the 'Transactions' contains a very elaborate paper by Sir Wm. Gull and Dr. Sutton in which the authors, while giving me some credit for having directed attention to thickening of the arteries, dissent entirely from my interpretation of the phenomena, and propound doctrines of great novelty and interest relating to the pathology of Bright's disease. In the present communication, I propose to inquire whether these novel doctrines are consistent with known pathological facts and established physiological principles.

The authors give a brief summary of the conclusions at which they have arrived at pp. 295-6 of their paper. The chief novelty to which they lay claim as a discovery is the doctrine that a "hyalin-fibroid" change in the minute arteries and capillaries in various tissues is the primary and essential phenomenon in cases of chronic Bright's disease with contracted kidney. This degeneration of the blood-vessels, they say, commonly begins in the kidney but it may begin in other organs; so that hypertrophy of the heart with degeneration of the blood-vessels may be found associated with healthy kidneys, the contraction and atrophy of the kidney when present being but "part and parcel of the general morbid change."

The morbid state commonly called chronic Bright's disease with contracted kidney is believed by Sir Wm. Gull, and Dr. Sutton to be essentially an "arterio-capillary fibrosis," the morbid changes being allied to senile changes, but "probably due to distinct causes not yet ascertained." Before proceeding to examine their description of the hyalin-fibroid change in the blood-vessels and in the kidneys, I desire to direct attention to some difficulties which have been suggested to me by a perusal of their paper.

In order to prove that the cardiac and vascular changes are not a result of the kidney disease they refer, at p. 286 (vol. lv), to three cases in which the kidneys were "little contracted" but the minute arteries and capillaries were much thickened by "hyalin-fibroid" substance. On turning to these cases I find that case 7, at p. 300, was that of a man,

forty-two years of age who had very emphysematous lungs, with capillary bronchitis; case 10, at p. 303, was sixty-nine years of age and died of senile gangrene, and case 20, at p. 309, was that of a man, æt. 51, who had vegetations on the aortic valves, with dilated heart's cavities and hypertrophied walls.

The cardiac changes in these three cases are supposed by the authors to be a result of hyalin-fibroid degeneration of the minute arteries and capillaries. I would suggest, however, that these changes might with more reason be referred in the first case to the emphysematous lungs with obstruction, primarily of the pulmonary, and secondarily of the systemic circulation; in the second case, to degeneration of the walls of the larger arteries resulting in senile gangrene; and in the third case to incompetence of the aortic valves, which is usually deemed sufficient to explain dilatation and hypertrophy of the heart.

In further proof of their doctrine that the arterio-capillary change is the primary and essential condition they refer again, at p. 286, to cases in which the kidneys were healthy while the heart was much hypertrophied.

Three cases are given in illustration of the co-existence of cardiac hypertrophy with healthy kidneys. Case 2, at p. 297, was that of a woman sixty-three years of age, who, therefore, may probably have had senile degeneration of her large arteries. The heart was heavy, but, death having resulted from rupture of the left ventricle, its muscular tissue must have undergone other changes than hypertrophy; and kidneys weighing fifteen ounces could scarcely have been healthy, even though their surface was "almost smooth." Case 3, at p. 297, was a man, seventy-seven years of age, whose hypertrophied and dilated left ventricle may probably be referred to senile changes in the larger arteries. And, lastly, case 19, at p. 308, was that of a man, aged sixty-two, who died suddenly comatose. His cerebral arteries were very atheromatous, and his lungs were emphysematous. In the condition of the larger arteries and lungs, we have, I think, a sufficient explanation of the dilatation and hyper-

trophy of the left ventricle, but, in addition, we find that the kidneys weighed only eight ounces; yet we are told that "with the exception of the capsule being adherent they were normal." We find then that one case in which the kidneys weighed fifteen ounces and another in which their weight was only eight ounces are set forth as examples of cardio-vascular changes with healthy kidneys. This implies that the kidneys of adults may differ in weight in the proportion of nearly two to one and yet be quite sound in structure.

While the authors refer to cases such as these to prove that hypertrophy of the left ventricle occurs unconnected with renal disease, they maintain that there is a constant association between the cardiac hypertrophy and the hyalin-fibroid change in the minute arteries and capillaries, and they state that there is a direct relation between the cardiac and the vascular change (see pp. 289 and 290). Yet it would seem that this pathological law is not without exceptions, for we find it stated at p. 292, par. 2, that "in a few cases the vessels of the pia mater were thickened by this 'hyalin-fibroid' change whilst the kidneys and heart were healthy."

Their explanation of the manner in which the vascular change causes the cardiac hypertrophy is the following (p. 290, par. 3):—"The hyalin-fibroid material in the walls of the arterioles must be an impediment to elasticity, and it can be experimentally shown that greater force is required to propel a fluid continuously through a non-elastic than through an elastic tube. The left ventricle, therefore, owing to this diminished elasticity of the arterial walls has, of necessity, to contract with greater force to carry on the circulation."

Now, upon this statement I beg to remark, that the explanation of the hypertrophy of the left ventricle is clearly a physiological one; the hypertrophy is said to be a result of the necessity for more forcible contraction to carry on the circulation. Yet the authors, at p. 285, referring to my explanation of the changes in the heart and arterioles as a physiological result, express their belief that "the cardio-vascular changes are throughout a morbid one." It appears,

however, that the authors of this criticism agree with me that the hypertrophy of the left ventricle is a physiological result of excessive muscular contraction called forth by some impediment in the course of the circulation; we differ only as to the cause of the impediment; and I submit that the explanation which I just now quoted from their paper is inconsistent with the facts of anatomy and with the doctrines of physiology.

In that explanation they appear to confound the elasticity of the larger arteries with the muscularity of the smaller vessels. The elastic resiliency of the larger arteries assists in propelling the blood onwards, and converts the intermitting jet from the heart into a continuous stream in the minute arteries and capillaries. The loss of this elasticity, therefore, by degeneration of the arterial walls, involves a loss of propulsive power, and thus imposes extra work on the heart. Hence, the common occurrence of hypertrophy of the left ventricle as a result of senile degeneration of the large arteries. On the other hand, the muscular contractility of the minutest arteries is a force which antagonises the heart and the larger elastic arteries; so that atrophy and degeneration of the muscular walls of the minute arteries tends to impair not the propelling but the retarding forces concerned in the circulation; and such degenerative changes would involve not an increase but a decrease of resistance to the blood-stream; unless the minute arteries and capillaries had their walls converted into rigid tubes with constricted canals, of which changes in the minute vessels I shall presently show that we have no proof.

The authors state, at p. 295, vol. lv, that the "hyalin-fibroid" change in the arterioles is attended with "atrophy of the adjacent textures in whatever organ it occurs." But I find it difficult to reconcile this statement with another statement which appears at, p. 287, par. 2, to the effect that "the minute arteries *in the walls of the heart* have been found much thickened by the formation of the 'hyalin-fibroid' substance;" and yet the walls of the heart are not *atrophied* but *hypertrophied*. Again the doctrine that

atrophy of adjacent tissues is an invariable result of thickening of the arterial walls is inconsistent with the fact that this thickening occurs in the minute renal arteries, not only in the small granular kidney, but also in the large smooth kidney of chronic Bright's disease. Obviously, then, thickening of the arterial walls is not, as the authors assert, invariably associated with atrophy of the adjacent tissues.

I now proceed to discuss the nature of those changes in the walls of the blood-vessels to which the authors of the paper give the name of "arterio-capillary fibrosis." When their paper was read before the Society, after hearing the description of the appearances in the walls of the vessels and examining the drawings, but before seeing their specimens, I ventured to suggest that the so-called "hyalin-fibroid" change was simply a result of the external fibrous coat of the artery being distended and rendered translucent by the fluid in which the specimens were preserved, that fluid being, as stated by the authors, a mixture of glycerine and camphor water (p. 280).

An examination of their specimens confirmed my first impression, and my subsequent inquiries have thoroughly convinced me that the hyalin-fibroid appearance is not an indication of a pathological change occurring during life, but a post-mortem physical result of the imbition of fluid by the coats of the vessels. I am indebted to the courtesy of Dr. Sutton for the opportunity of examining a number of his specimens, and I learnt from him that they were all mounted in the glycerine fluid by an assistant before they were examined microscopically by any one.

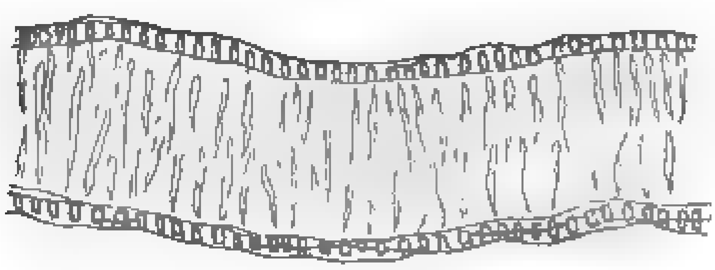
The authors, therefore, up to the time of reading their paper, had had no opportunity of comparing the appearance of the vessels when recently removed from the body with that which they assume after immersion in glycerine. This comparison I have made in a number of instances, and the following is the result.

1. I have never yet seen the hyaline thickening of the external fibrous tunic in vessels examined immediately after removal from the body.

2. The appearance is very common, but not constant, after the vessels have been immersed in a mixture of glycerine and camphor water. I have different specimens of arteries from the same subject. Some mounted in the glycerine fluid are conspicuously hyaline, while others preserved in weak spirit and water or in a solution of common salt of sp. gr. 1030 present a perfectly normal appearance. The solution of salt, however, is not sufficiently antiseptic to preserve the specimens for more than a few days.

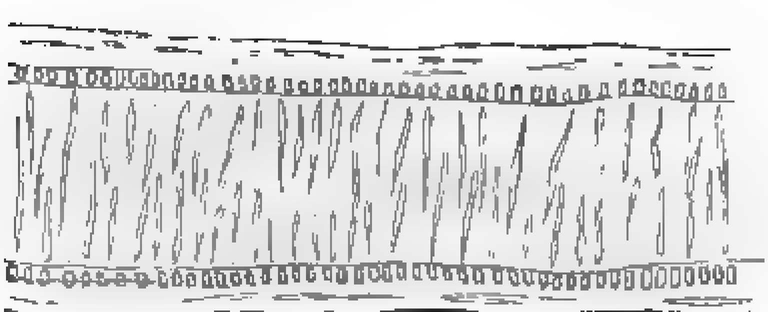
3. The glycerine fluid produces the hyaline thickening of the minute arteries of the pia mater after death from various diseases, having no relation to Bright's disease, and not being associated with hypertrophy of the left ventricle. Thus, for example, I have specimens of pia mater arteries from a woman who died of diabetes at the age of forty, and whose heart weighed only 6¾ oz. (see figs. 1 and 2). From another

FIG. 1.



Normal artery from the pia mater, mounted in dilute spirit. × 200.

FIG. 2.



Artery from the same pia mater as No. 1; but the fibrous tissue has been distended and rendered hyaline by the glycerine and camphor water in which it is mounted. × 200.

woman, æt. 40, who died of cancer of the ovary, whose heart weighed 8 oz. From an infant, aged eleven months, who, having had a cough for a few days, died suddenly, and probably from spasm of the larynx. The kidneys, heart, and all the organs were quite healthy, but the arteries of the pia mater, preserved in the glycerine fluid, present the "hyalin-fibroid" change very conspicuously.

I have also specimens of pia mater arteries from a boy, æt. 15, who had been in good health until he was attacked by typhoid fever, during the course of which he died from perforation of the bowel. There was no *post-mortem* evidence of disease except such as resulted from the fever. In particular the heart and kidneys were healthy. Portions of pia mater have been preserved in three different fluids; 1st, in dilute spirit; 2nd, in salt and water, sp. gr. 1030; and, 3rd, in equal parts of glycerine and camphor water. In Nos. 1 and 2 the vessels appear quite normal; in No. 3 the fibrous tunic is much distended and extremely hyaline. These specimens are on the table for inspection this evening.

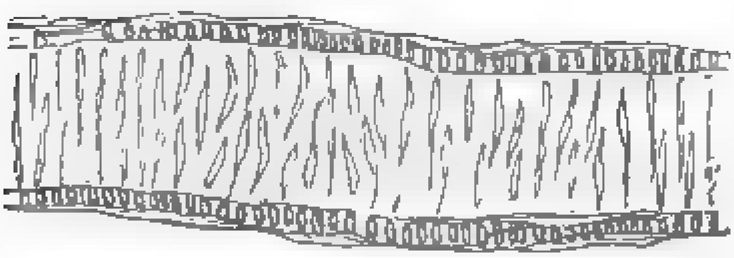
With regard to the physical conditions which favour the imbibition of fluid by the fibrous tunic of the arteries it is certain that while the process is favoured and quickened by acidity of the fluid it is prevented by its alkalinity.

The following experiment affords a good illustration of this principle:—Immerse a small piece of pia mater in water, or in glycerine, slightly acidulated by acetic acid; the arteries in a few seconds assume the hyaline appearance; then neutralize the acid by liquid ammonia, and immediately it will be found that the fibrous coat has shrunk, losing its hyaline character and presenting the fibrous wavy outline which it had before it was distended by the acid fluid. I have specimens of normal arteries from the same subject, one set preserved in the acid fluid, having the fibrous tunic distended and hyaline; the others, after being distended by the acid, have been made to shrink and again assume the fibrous character by the addition of ammonia; and in that condition they are preserved (see figs. 3 and 4).

FIG. 3.

Normal artery from the pia mater, the fibrous tunic distended and rendered hyaline by dilute acetic acid. × 200.

FIG. 4.



Artery which, after immersion in dilute acetic acid, and thus having been rendered hyaline like No. 3, has had its fibrous coat again contracted by the addition of ammonia. × 200.

This experiment shows that the appearance of the minute arteries may be rapidly and greatly changed by chemical conditions, which would be influenced by various circumstances, including, amongst others, probably the mode of death and the time after death at which the vessels are examined.

I look upon it as certain that the "hyalin" appearance so often referred to is always the result of post-mortem changes in the arteries. I believe that the appearance in question has never been seen in vessels recently removed from the body, and not subjected to artificial agencies; though it is possible that the change might occur, in the vessels of the pia mater, for instance, as a result of post-mortem maceration in the subarachnoid fluid.

It is incumbent on those who believe that the appearances described are the result of ante-mortem pathological processes to demonstrate them in vessels recently removed from the body and unchanged by artificial agents.

The glycerine fluid while distending the fibrous tunic often presses the muscular coat more or less irregularly inwards, so as to narrow the canal of the artery; frequently, too, it appears, as it were, to fuse together the muscular elements, rendering them indistinct and giving the thickened wall of the artery a homogeneous or a granular appearance. In other specimens the distension of the arterial wall appears to separate the muscular elements to a greater distance from each other (see Plate VI, fig. 3, in vol. lv). These changes are supposed by the authors of the papers to indicate atrophy of the muscular coat of the artery. I believe them to be simply due to post-mortem physical changes.

I should not now select as good examples of hypertrophy such specimens as are represented in figs. 2 and 3, Plate I, appended to my paper in the fifty-first volume of the 'Transactions.' In these specimens the muscular tissue has been rendered indistinct by the imbibition of glycerine. The appearances here described are quite distinct from the fatty degeneration of the walls of the vessels resulting from senile degeneration, and quite different from the waxy or lardaceous degeneration of the arterial walls.

In some of the arteries the fluid, passing between the middle and inner coat of the vessel, separates them from each other and gives that appearance which the authors describe and figure as a "thickening of the tunica intima." This appearance is shown by the authors in figs. 3 and 4, Plate V, vol. lv.

Again, they affirm that the thickening extends to the walls of the *capillaries;* it is an "arterio-capillary fibrosis." Here, again, I am compelled to dissent. I have seen no capillaries thickened, except the Malpighian capillaries of the kidney; and that, in cases of Bright's disease, is probably a result of the continued transudation of albuminous and fibrinous materials through their walls. I have seen no thickening of

the capillaries in the pia mater. Fig. 2 in Plate VI, which is described as a thickened capillary, I take to be an artery thickened and transformed by glycerine. Fig. 1, in the same plate, with transparent oval bodies in the walls, is unlike any vessel that I have seen, and extremely unlike any capillary of the pia mater.

I do not think it necessary or desirable to criticise at length the author's account of the minute anatomy of the contracted kidney. The sum and substance of their description is that "hyalin-fibroid" material is found here, there, and everywhere in the kidney.

In the year 1847 two papers on renal pathology were read before this Society and published in the thirtieth volume of the 'Transactions.' Mr. Simon was the author of one of these papers and I contributed the other. Both papers were read the same evening; in both papers the same appearances were described and figured, but Mr. Simon and I differed essentially in our interpretation of the phenomenon. The appearances which Mr. Simon described as microscopic cells and cysts I interpreted as tubes denuded and rendered transparent by the destruction and removal of their gland-cells. I have since seen no reason to doubt that my explanation of the appearances in question was the true one, or that the description then given of the minute anatomy of the contracted kidney was, in the main, correct. The appearances which then formed the subject of controversy and which may readily be demonstrated in the contracted Bright's kidney are completely ignored or lightly passed over by those pathologists who make unsatisfactory attempts to explain the structural changes by hyperplasia of connective tissue, or hyalin-fibroid deposit between the tubes.

The description which Sir William Gull and Dr. Sutton give of the appearance presented by the renal arteries in the contracted kidney is so remarkable that it demands especial comment. Fig. 7, in Plate VI, represents a transverse section of a thickened renal artery, and in the description of the plate the artery is said to be "greatly

thickened by hyalin-fibroid changes in the outer layer of the vessels."

The surface of the section shows two distinct layers of fibres, an inner longitudinal and an outer circular, both of which are *muscular*, as may be clearly seen in many specimens in my possession. Sir William Gull and Dr. Sutton, however, assume that the inner longitudinal layer alone is muscular, and at p. 274 (par. 6) they say that "external to the muscular nuclei there was a quantity of hyalin-fibroid substance, and the layer formed by this material was much thicker than the muscular layer" (Plate VI, fig. 7, in vol. lv). Again, they say (at p. 278, par. 4), "Where the kidney disease was far advanced hyalin-fibroid changes were seen in the minute renal arteries precisely similar to those observed in the arterioles of the pia mater and of other parts of the body." It is evident, therefore, that they believe the outer layer in the renal artery to be the counterpart of the so-called "hyalin-fibroid" layer in the arteries of the pia mater. The fact being that this layer in the pia mater arteries is external to the circular muscular fibres, and is composed of the distended fibrous tunic; while in the renal artery the fibrous tunic is inconspicuous and that which they mistake for it is the circular layer of muscular fibres.

In my first published account of the hypertrophied renal arteries in cases of chronic Bright's disease ('Med. Chir. Trans.,' vol. xxxiii) I described and figured the inner longitudinal and the outer circular layer of muscular fibres. The appearances described are quite constant; they were well seen in some of the author's specimens exhibited at the conversazione in June, and were clearly represented by their artist; yet they have been misinterpreted in the manner described. The existence of hypertrophy of the muscular walls is denied and the hyalin-fibroid change is supposed to explain the phenomena.

At the foot of page 277 we find, in a note, the following statement :—

"Recent observations on the minute anatomy of the

kidney raise a doubt whether casts formed in the convoluted tubules* of the cortex can escape."

It would be interesting to know what observations have led to this curious piece of scepticism. No one who has studied the microscopic diagnosis of renal diseases can, for a moment, doubt that casts from the convoluted tubes of the cortex continually escape and are found in the urine. If this were not so the various forms of tube-casts would throw no light upon the state of the cortical portion of the kidneys, which is acknowledged to be the seat of Bright's disease. We find, however, that these casts from the cortex sometimes retain their convoluted form even after passing through the straight tubes. They often contain epithelial cells which, from their characteristic appearance, are known with absolute certainty to have come from the cortex and not from the tubes of the cones. And, lastly, in some cases of hæmaturia, blood which has been poured into the very extremities of the convoluted tubes from ruptured Malpighian capillaries, escapes and appears in the form of blood casts in the urine.

To doubt the possibility of the blood passing from the right to the left side of the heart through the lungs would be scarcely more unreasonable than to doubt the escape of casts from the convoluted tubes of the kidney in most cases of Bright's disease.

The authors object to my doctrine that the hypertrophy of the left ventricle, in cases of chronic Bright's disease, is a consequence of the increased resistance to the circulation occasioned by excessive contraction and resulting hypertrophy of the minute muscular arteries. They say truly that hypertrophy of the heart, although usually present in the advanced stages of the contracted kidney, is absent in a large proportion of cases of large white kidney.

My belief is that when, in cases of chronic Bright's disease with either large or small kidneys, the left ventricle is hypertrophical without disease of the valves or of the

* In the note referred to, "convoluted *tubercles*" is evidently a misprint for "convoluted *tubules*."

large arteries to explain it; in all such cases the minute arteries throughout the body, but especially in the skin and mucous membranes, will be found to have their muscular walls hypertrophied. I have as yet met with no exception to this rule. In explanation of the common occurrence of hypertrophy of the left ventricle in connection with the contracted kidney and its frequent absence in cases of large white kidney, I beg to suggest the following considerations :

1st. With the contracted kidney there is, as a rule, absence of dropsy or but little dropsy, a free secretion of water by the kidney, with retention of urinary solids in the blood; and this uræmic condition is, in various ways, the cause of death. On the other hand, in cases of large white kidney, there is a more scanty secretion of water, and, in consequence, a state of hydræmia and dropsy. In the first class of cases the urinary solids accumulating in the blood probably excite the contraction of the systemic arterioles, and thus directly cause their hypertrophy and indirectly that of the left ventricle. In the second class of cases—those associated with large white kidneys—while there is some retention of urinary solids there is a disproportionate accumulation of water, by which the contaminated blood is diluted and thus rendered less irritating to the minute arteries.

This is probably, in part at least, the explanation of the fact that the arterial resistance is less and the cardiac hypertrophy less constant and pronounced in cases of large white kidney with consequent hydræmia than in cases of contracted kidney with resulting uræmia.

2nd. But there is yet another class of facts to be taken into account. In connection with the lardaceous form of disease in the kidney and in other organs there is usually a widely spread tendency to degeneration of the small arteries. The degenerative changes affecting the muscular walls impair their contractile power, and the result is not an increase but a decrease of resistance in the terminal arteries. The peripheral resistance which calls forth increased contraction with resulting hypertrophy of the left ventricle is wanting, and in these circumstances there is absence of cardiac

hypertrophy. It appears to me that these apparently exceptional cases prove the rule; namely, that there is, in the absence of valvular lesions and senile degeneration of the walls of the large arteries, a direet and constant relation between hypertrophy of the left ventricle of the heart and hypertrophy of the muscular walls of the minute arteries throughout the body. The occasional, though rare, occurrence of fatal cases of contracted kidney without cardiac hypertrophy is not inconsistent with the theory that when they coexist they stand to each other in the relation of cause and effect. Cases of large white kidney sometimes run their course without the occurrence of dropsy; but we do not, therefore, deny or doubt that the dropsy which is frequently associated with this form of disease is a result of the blood changes induced by the renal disease.

It is stated at p. 289 of the author's paper "that in eighteen cases the left ventricle was found hypertrophied without valvular disease or pericardial adhesion to explain it, while the kidneys were healthy excepting that they were slightly granular, or, in other words, 'coarse' and contained some cysts." It is therefore inferred that in these eighteen cases the hypertrophy was "the older and preceding condition." Upon this statement I would suggest—1st. That a minute examination of the kidneys might have shown more advanced degeneration than appeared upon the surface; and, 2nd, it is not unlikely that in some of these cases the cardiac hypertrophy was, in part at least, explained by degeneration of the walls of the larger arteries, as in some of their cases occurring in old people to which I have before referred.

The atheromatous degeneration of the large arteries, which is often associated with chronic Bright's disease, may be in part, perhaps, explained by the noxious influence of contaminated blood, and in part by the undue strain to which the arteries are subjected between the two opposed forces, that of the hypertrophied left ventricle behind and that of the resisting muscular arterioles in front.

I need not here repeat my objection to the author's

doctrine that hypertrophy of the left ventricle is always associated with and explained by the hyalin-fibroid change in the minute arteries; this change being, as I believe, not a pathological change at all but the result of artificial influences operating after death.

The statement that the morbid changes included under the head of chronic Bright's disease with contracted kidney are the result of "causes not yet ascertained" is not in accordance with the results of my own observation.

There are few cases of this form of disease which are not traceable to some probable cause. Amongst the most common causes is the excessive consumption of food and of alcoholic stimulants either with or without the association of decided gouty symptoms; but I have seen many cases in which, as I believe, this form of renal disease has resulted from chronic dyspepsia in persons of strictly temperate habits. The one condition which is common to all these cases is that the degeneration of the kidney is directly caused by the excretion of abnormal and irritating materials, the products of faulty digestion; more commonly associated with habitual excess of food and stimulants, but not rarely without such excess.

I have always maintained that Bright's disease, in all its forms, may be traced back to some anterior morbid change in other tissues and organs, the proximate cause of the renal disease in every case being the contamination of the circulating blood by abnormal products, which the kidney, in the discharge of its excretory function, is called upon to eliminate.

The doctrine that the area of the morbid state under discussion "may be said hypothetically to correspond to the area vasculosa" is too transcendental for everyday use, and it is not in accordance with the fact that the morbid changes are, as a rule, limited to definite portions of the arterial system, while the veins and capillaries, excepting only the Malpighian capillaries, are not implicated. The term "arterio-capillary fibrosis" appears to be a misnomer, for the capillaries generally are unchanged, and if my interpre-

tation of the phenomena is correct there is no morbid "fibrosis" of the arteries.

In conclusion, I beg to express my thanks to Sir William Gull and Dr. Sutton for having so forcibly directed attention to the cardio-vascular changes in chronic Bright's disease. Differing as we do entirely in our interpretation of the phenomena we agree in the opinion that the subject is one of great interest and importance.

AN ANALYSIS

OF

SHIP AIR AND ITS EFFECTS.

BY

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COMMUNICATED BY

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To isolate and clearly define those adverse agencies which oppose health and longevity is obviously the first step towards their amelioration or removal. Although the vitiation of ship air has been long recognised, and ventilation is not unattended to either in the construction or after management of ships, the following observations will go far to show that their ventilation is still imperfect, and ship air more impure than commonly supposed. We know exactly the contamination of what the landsman and soldier breathe, and ventilate their abodes accordingly. But we have yet little else to indicate how much, or by what, ship air is polluted, beyond such unsatisfactory phrases as foul, close, &c. Facts form the surest criterion of where and to

what extent hygienic reform is necessary. Without an analysis we are working, so to speak, in the dark. To prove how vitiated ship air may become, and thereby show how necessary thorough ventilation is, is therefore an important preliminary toward giving seamen a healthier atmosphere.*

Ship air is more impure than barrack and house air, because—

I. Immersion complicates ship ventilation; preventing it below and often at the sides.

II. Many handicrafts are unavoidably carried on and stores kept, so to speak, in the midst of the crew.

III. The men are berthed and stores kept in the centre and lower parts, *i. e.* those most difficult to ventilate.

IV. And chiefly from overcrowding. While the soldier has, at least, 600 cubic feet of sleeping room, and from 1500 to 1800 in the tropics, an increase to 3000 feet being recommended,† Table I, giving the average air space and distribution of the men in a frigate, the type of a large part of H.M. and other navies, shows—

1st. That although the uncurtailed internal capacity would give her crew of 600 an average of 576 cubic feet per man, the crew proper really have no more than 63 on the densely crowded lower deck at meals, and from 105 to 222 at night on the sleeping decks where the hammocks usually touch.

2nd. That each cadet has only 100 cubic feet at meals, 117 at study, and from 242 to 506 at night.

3rd. That each ward-room officer has only from 300 to 400 cubic feet.

4th. That the allotted space is the same in all climates.

* Cannot the barometer, and other apparatus for indicating the physical condition of the air, be supplemented by some instrument to show at a glance the degree and nature of its impurity, without having recourse to the more difficult and tedious processes of analysis?

† Parkes, 'Practical Hygiene.'

TABLE I.—*Distribution of men and average air space* (H = *harbour*; S = *sea*).

Place.		Number of men. Night.	Number of men. Day.	Total cubic space in feet. Night.	Total cubic space in feet. Day.	Average cubic space per man in feet, corrected for bodies and bedding. Night.	Average cubic space per man in feet, corrected for bodies and bedding. Day.
Entire ship		H 600	H 600	H 345,637	H 345,637	H 576	H 576*
		S 600	S 600	S 345,637	S 345,637	S 576	S 576
Lower deck tier †	Before steerage	H 300	H 500	H 31,548	H 29,948	H 105	H 63
		S 200	S 500	S 32,348	S 29,948	S 161	S 63
	Steerage (cabins included)	H 44	.	H 14,676	...	H 333	...
		S 44	...	S 14,676	...	S 333	...
	Cockpit	H 8	...	H 2,053	...	H 257	...
		S 8	...	S 2,053	...	S 257	...
	Ward room	H 7	H 30	..	H 3,505	...	H 116
		S 7	S 30	...	S 3,505	...	S 116
	Chest room	H 26	..	H 6,700	...	H 242	...
		S 26	..	S 6,700	...	S 242	...
	Lower deck (entire)	H 285	H 600	H 66,709	H 64,989	H 237	H 108
		S 385	S 600	S 66,709	S 64,989	S 173	S 108
Main deck tier	In front of office	H 192	...	H 41,251	..	H 222	...
		S 292	...	S 40,451	...	S 138	...
	Half deck (cabins included)	H 15	...	H 11,760	...	H 784	...
		S 15	...	S 11,760		S 784	...
	Study	H 8	H 34	H 4,049	..	H 506	H 117
		S 8	S 34	S 4,049	..	S 506	S 117
	Gun room (cadets)	H 0	H 72	At meals.	H 7,248	...	H 100
		S 0	S 72		S 7,248	...	S 100
	Main deck (entire)	H 215	...	H 64,404	...	H 299	...
		S 315	...	S 63,604	..	S 201	.
Special parts	Store rooms	H 0	...	H 0	...	S 480	...
		S 8	...	S 3,830	.	...	.
	Cells	H 1	..	H 120	H 120	H 120	H 120
		S 1	...	S 120	S 120	S 120	S 120
	Cabins on lower deck	H 1	...	H 300	...	H 300	...
		S 1	..	S 300	...	S 300	...
	Cabins on main deck	H 1	...	H 432	...	H 400	...
		S 1	...	S 432	...	S 400	..

* If corrected for engines, stores, &c., these averages would be reduced to 450 cubic feet.

† This term corresponds with floor or flat, and must not be confounded with the hold "tiers," where spare sails, &c., are kept.

The usual impurities of ship air are of animal, vegetable, and mineral origin, and comprise odorous and inodorous gases, vapours, and solids, all more or less unhealthy, and derived—

1st and chiefly, from the skin, lungs, &c., of the crew, which yield carbonic acid, ammonia, sulphuretted hydrogen, volatile organic matter, watery vapour, &c.

2nd. From the ship,—organic and inorganic odours, and minute particles of wood, paint, cordage, whitewash, leather, iron, brass, &c.; from the bilge and lining—ammonia, sulphide of ammonium, sulphuretted hydrogen, and occasionally microscopic animal and vegetable organisms.

3rd. From the stores,—particles of bread, cotton, wool, &c.; from the slow combustion of coal in the bunkers, perhaps carbonic acid and carbonic oxide; from the rapid combustion of coal, candles, oil, &c., these gases and soot, sulphurous acid, sulphuretted hydrogen, ammonia, sulphide of ammonium (Parkes); from cooking,—volatile, animal, and vegetable odours.

4th. From gun firing,—carbonic acid, carbonic oxide, hydrogen, sulphuretted hydrogen, suspended salts (Parkes).

5th. From sea spray,—salts, and moisture.

The following analysis of the air of the wooden screw-frigate Bristol (ventilated chiefly on the old windsail plan), made during a four months' voyage (July to November, 1871) from the Cape of Good Hope to England, represents that of a class of ship still large and likely to remain so. In modern ironclads the impurity probably does not differ much from this either in nature or extent. Less sulphuretted hydrogen, doubtless, emanates from an iron than from a wooden bilge; but other impurities may be added, and the chief ones, carbonic acid and organic matter, are probably not materially if at all lessened. Because, although greater size and comparatively smaller crews give a larger average air space, and modern ventilation favours purity, they have smaller ports, are altogether more closed in, and thus admit pure and emit foul air less freely; especially deeply immersed ships of the turret-and-ram class.

The degree of impurity doubtless varies. In some ships it may be more, in others less than this. But there is reason to believe that in all it is higher than usually suspected, and than it ought to be. In those merchant vessels in which the men are dirtier, more crowded, and the ventilation bad, the vitiation is doubtless still greater.

1. *Carbonic acid.*

The results of 150 analyses prove that this is the most abundant and important impurity; and Table II of 111 by Pettenkofer's test* shows—

1st. That ships throughout are, at all times and in different degrees of crowding, pervaded and, doubtless to a certain extent, enveloped by an atmosphere containing from 4·20 to 33·71, average 14·60 volumes of CO_2 per 1000,† the oxygen in the air being, of course, correspondingly diminished.

2nd. That this gas is most abundant, and varies from 4·20 to 28·85 volumes, when and where there is most crowding, *e.g.* on the lower deck and middle tiers, at meals and by night; proving that respiration, pulmonary and cutaneous, is its chief source.

3rd. That owing to diffusion, direct vitiation from combustion, the bilge, &c., and difficult, therefore imperfect, ventilation, the lower parts generally, show from 9·34 to 26·37 volumes.

* The size of the glass bottle employed was 215 fl. oz. (or 372 cubic inches), an ordinary bellows being used to charge it. The solution of oxalic acid to test the aqua calcis before and after its contact with the air was of the usual strength, viz. 2·25 grammes to one litre of water. The aqua calcis usually contained from 20 to 30 milligrammes per 30 cubic centimètres. The strength mentioned by Parkes (34 to 39 milligrammes) is more apt than a weaker one, from change of temperature and evaporation, to leave a deposit of hydrate of lime on the sides of the bottle, thus interfering with results. The lime water and air were kept in contact from ten to twelve hours. The subsequent calculation was that detailed in Parkes's 'Practical Hygiene.'

† This and other gases are reckoned throughout at so many volumes per 1000.

4th. That, both from direct vitiation and diffusion, the more open, better ventilated, and less crowded main deck tier contains from 4·20 to 16·32 volumes.

5th. That, for special reasons, the air is sometimes very impure. Thus, the crowded ward room pantry showed 19 volumes, the confined ill-ventilated store rooms which drew air second hand from the lower deck from 13·29 to 19·34 volumes, and the thrice vitiated air of the small worse ventilated cell or prison, derived from the store rooms, from 19·80 to 33·71 volumes.

6th. That officers and men breathe air nearly equally impure: ward room cabins averaging 14·49 volumes; main deck ones 10·27 volumes; the well-ventilated but crowded cadets' mess room 7·86 volumes; their study 15·42, and, in rough weather, when barred in, 27·61 volumes; their sleeping room below 16·62 volumes.*

* First wonder at the great impurity of ship air will cease, like my own, when the conditions are considered, especially the limited air space, lengthened crowding, and ventilation.

TABLE 2.—*To contrast the CO_2 in the air of different parts.*

		Number of analyses made.	Volume of CO_2 per 1000 of air at different times of the night and day and different degrees of crowding. Highest.	Lowest.	Averages.	
Upper tier.	Main deck	10	16·3	7·83	11·04	11·71
	Half deck	1	13·99	13·99	13·99	
	Study	3	27·61	5·59	15·42	
	Mess room	2	11·53	4·20	7·86	
	Main deck cabins	2	12·05	8·49	10·27	
Middle tier.	Lower deck	47	28·06	4·20	16·33	15·86
	Steerage	1	10·58	10·58	10·58	
	Ward room	4	22·14	7·70	17·82	
	Chest (cadets' sleeping) room	7	28·85	10·01	16·62	
	Ward room and steerage cabins	2	16·23	12·76	14·49	
	Do., pantry	1	19·34	19·34	19·34	
Lowest tier.	Stokehole	15	26·37	4·54	14·24	15·34
	Engine room	2	13·99	12·67	13·33	
	Screw alley	2	11·21	9·34	10·22	
	Cockpit	2	14·96	14·72	14·84	
	Store room	3	19·34	13·29	16·42	
	Cells	7	33·71	19·89	23·01	
	Total	111	...	...	...	
	Highest	...	33·71	...	...	
	Lowest	...	...	4·20	...	
	Average	...	...	...	14·60	

These figures show a great departure from the ·4 volumes per 1000 (Roscoe) of the pure external air, alone fit for respiration,* and large excess over the ·600 volumes in our best, the 1·98 volumes of our worst ventilated barracks (Parkes, Roscoe, Fyffe de Chaumont); and the ·8 to 1 volumes in well ventilated private houses (Œrtel). And they are more akin to the 3·1

* The quantity of CO_2 in pure sea air has not yet been determined. It is more difficult than might be supposed to charge a bottle with air completely out of the range of emanations from the ship. Analogy would lead us to suppose, however, that it is less than in land air.

volumes (Roscoe) in boys', and 7·23 volumes (Pettenkofer) in girls' schools, or the 3·65 volumes (Roscoe) and 10 volumes (Dalton) of crowded meetings. The smaller impurity in schools and assemblies than in ships arises from the crowding being neither so great nor prolonged, the air space greater, and the usually more perfect ventilation. In schools the lungs are smaller, but this and the carbon elimination are, perhaps, counterbalanced by the quicker respiration.*

So also according to the crowding and renewal of air the CO_2 may vary even on opposite sides of the same deck; thus—

Main deck	Starboard (weather) side	9·26 volumes per 1000
	Port (lee) side (fewer sleepers and better ventilated)	7·83 " "

For an obvious reason the CO_2 is also less near open ports, scuttles, hatchways, windsails, &c.

The CO_2 issuing from the lungs and skin of the men sleeping close to the beams overhead does not gravitate towards the deck, but is more or less rapidly diffused. Thus, on the lower deck at night (temp. 80° F.) the quantity in one experiment was identical in both places, viz. 7·8 volumes. On a second occasion, however (temp. 76° F.), it was densest near the deck by 0·71 volumes. The streams of pure air entering by many different channels, and the more numerous ones of impure air from the lungs, evidently do not mingle at once. This corresponds with the experiments of Lassaigne, Pettenkofer, Roscoe, and Gore. Moreover, the whitewash with which the interior of ships is often painted is usually found, after six weeks or two months, especially on the lower deck

* In connection with the impurity of ship air, it will be also useful to remember—

1st. That air containing more than 1 volume of carbonic acid per 1000 is unfit for *continued* respiration (Roscoe).

2nd. That expired air contains from 30 to 60 vols. (Taylor), and never more than 100 vols. (Allen and Pepys).

3rd. That air containing from 100 to 120 vols is speedily fatal to man (Taylor).

4th. That air containing from 120 to 150 vols. will extinguish a taper (Taylor).

between the beam, *i.e.* near the heads of the sleepers, to be highly effervescent under acids, and almost wholly converted into carbonate, proving that, altogether apart from its cleanliness and disinfecting properties (Angus Smith), frequent whitewashing with slacked lime is highly sanitary, because absorbent of CO_2, and doubtless organic matter and watery vapour also.

The following table, regarding the lower or mess deck at sea, shows—

1st. That the CO_2 varies by night and by day, rising and falling with the crowding; and is great, *e.g.* at breakfast, 6½ a.m. (20·65 volumes), dinner, 12½ p.m. (25·74 volumes), and supper, 5½ p.m. (17·80 volumes), culminating at 7½ p.m. (26·53 volumes) when the men are mostly below and the lights lit.

2nd. That it remains high during night (14·98 to 19·54 volumes) when the ship is most shut in, and the ventilation often least attended to. Roscoe's experiments, showing that the lungs, especially of working men, absorb most oxygen by night, the blood storing it up for the following day, makes the great impurity of ship air during the long night watches, when the majority of the men are below and asleep, a fact of great hygienic importance.

TABLE 3.—*To contrast the CO_2 in lower deck air at different periods of the twenty-four hours.*

	Volumes of CO_2 per 1000 of air.
3 a.m.	14·98
6½ „	20·65
9½ „	14·24
10 „	18·24
Noon	18·45
12¼ p.m.	13·57
12½ „	25·74
3 „	15·6[illegible]
4½ „	9·38
5½ „	17·80
7 „	16·27
7¼ „	20·65
7½ „	26·53
11¼ „	19·54
11¾ „	[illegible]

Table 4 shows that crowding has a like effect elsewhere, *e.g.* in the officers' quarters.

TABLE 4.—*To contrast the CO_2 in different parts when empty and crowded.*

			Volumes of CO_2 per 1000 of air.
Ward room...	Empty	3 a.m.	7·10
	Full	7 p.m.	22·12
Gun room ...	Empty	3 a.m.	4·20
	Full	6½ p.m.	11·53
Cadet's study	Empty	3 a.m.	5·59
	Full	10 a.m.	27·61

The effect of the lower deck lights, *i.e. combustion* in increasing CO_2, as shown by table 3 (7½ p.m.), is still better illustrated by the boiler fires when steaming (Table 5), which made it rise in the stokehole from 12·87 volumes sailing, to 19½ under three, and 26·37 volumes under four boilers. It will presently appear that this added impurity is not local but generally diffused through the ship.

TABLE 5.—*To contrast the CO_2 in the stokehole under sail and steam.*

	Temp. of external air.	Volumes of CO_2 per 1000.
Sailing in S.E. trade wind	81° F. (shade) ...	12·87
Steaming in equatorial doldrums with three and four boilers lit ...	82° F. (shade) ...	19·55 to 26·37

As CO_2 is constantly forming, thus requiring constant removal, it necessarily varies with the *ventilation*. Thus, Table 6, contrasting it when at anchor and unavoidably much closed in during rough wet weather, and when better ventilated at sea by trade winds, &c, shows—

1st. That it was everywhere reduced in the second instance from an average of 20 to 12·32 volumes.

2nd. That the reduction was greatest where most required,

i. e., on the lower or mess deck, viz. from 24·18 to 9·78 volumes.

TABLE 6.—*To contrast the CO_2 under different degrees of ventilation.*

	Indifferent ventilation; Simon's Bay. Volumes of CO_2 per 1000.	Fair ventilation; S.E. trades near St. Helena. Volumes of CO_2 per 1000.
Main deck ...	12·09 ...	9·72
Lower deck ...	24·18 ...	9·78
Chest room ...	16·53 ...	12·55
Stokehole ...	16·64 ...	9·76
Bilge ...	20·22 ...	11·83
Store room ...	16·64 ...	12·73
Cells ...	33·71 ...	19·89
Averages ...	20·00 ...	12·32

The latter result was chiefly due to the rapid withdrawal of the foul air up the funnel by the agency of bogies in the boiler fires, in imitation of Baker's, Fanshawe's, and Edmonds' systems of outward suction by heat (nature's chief ventilating agent), largely employed in modern ventilation. This system, easy enough in sea-going naval ships, now invariably steamers, has been successfully applied* in H.M.S. "Britannia," a wooden three-decker, as it might in all similar harbour ships, by means of a large lit stove about the centre of the kelson, with a vertical funnel opening high above the upper deck.

The local and general purifying effect of small boiler fires, which create sufficient draught to carry off their own products along with the impure air, contrasts strongly with the vitiating effect of the larger ones employed for steaming (as shown by Table 5) which generate CO_2 and other noxious gases faster than they can be withdrawn by the funnel. On the other hand, for an obvious reason, the effect of open fires or bogies often wisely used to dry decks, &c., and sometimes erroneously to "burn up foul air," corre-

* In September, 1872. For description, see Reed's 'Naval Science' for April, 1873.

sponds to the latter rather than the former, inasmuch as they increase the impurity of ship air by adding CO_2 and even more noxious gases (SO_2, CO, &c.), which are not directly carried off, but diffused. Thus, on one occasion while lower deck air showed 10·82 volumes of CO_2, the usually pure engine room air, near two bright red bogies, had 12·67 volumes.

Any improvement or deterioration in the *ventilation*, resulting from the direction and strength of the wind, *i. e.*, the angle and force with which it strikes the ship, causes marked variations in the impurity of ship air. Thus, Table 7 shows—

1st (column 1).—That the CO_2 rose greatly throughout, *e. g.* on the lower deck to 18·59 volumes, in the sultry equatorial *calms*, from the external and the ship air being both almost stagnant.

2nd (column 2).—That when *steaming* in the same calms, it rose still higher, *e. g.* to 28 volumes on the lower deck, from the combustion of coal, and imperfect renewal of air.

3rd (column 3).—That it still remained high (*e. g.* 25 volumes on the lower deck) when riding or steaming *head to wind* during a breeze which glanced off the bows and sides, and that the foul air was driven aft, accumulating in the chest or cadets' sleeping room to 28 volumes.

4th (column 4).—That with the wind *abeam*, and fully entering her ports so as to blow the ship out, the CO_2 fell throughout, *e. g.* on the lower deck to 8·6 volumes, *i. e.* to one third of its former quantity.

5th (column 5).—That with the wind on the quarter or bow, *i. e. before or abaft the beam*, and, therefore, entering the ship imperfectly at an acute angle, the volume of CO_2 again rose, *e. g.* on the lower deck to 14·76 volumes.

TABLE 7.—*To contrast the CO_2 at* 11 *p.m, when*—1, *sailing;* 2, *steaming;* 3, *riding head to wind;* 4, *sailing, wind abeam;* 5, *at anchor, wind on quarter.*

	Volumes of CO_2 per 1000.				
	Column 1. Sailing in calms of equator. Temp. 81° F.	Column 2. Steaming in equatorial calms. Temp. 82° F.	Column 3. Barbadoes; riding head to wind. Temp. 80° F.	Column 4. Barbadoes to Bermuda; sailing side wind. Temp. 82° F.	Column 5. Bermuda; anchored, wind on quarter. Temp. 81° F.
Lower deck......	18·59	28·06	25·03	8·60	14·76
Main deck	16·23	16·3	...	...	...
Stokehole	12·87	26·37	23·64	...	...
Chest room......	20·69	...	28·25	10·01	...
Cells	23·15	...	25·96	21·8	...

As a rule, at least in fine weather, ship air is purer at sea than in harbour, because set sails materially aid ventilation, windsails, &c., act better, and the wind is usually stronger. But, as in harbour (Table 6, column 1) so at sea, a closed-in and badly ventilated ship, from rough weather, is followed by a great increase in the impurity of her atmosphere. Thus, near the Azores, from this cause, the CO_2 rose on the lower deck from 8·9 to 21·54 volumes.

Although ships, for coolness and comfort, are usually more opened out in the tropics, the preceding tables show that ship air, as a rule, is not purer there, because for several reasons it is more stagnant and less speedily diffused and renewed. There they generally sail *with* and nearly as fast as the wind, and there is less difference between the internal and external temperature to favour natural ventilation.

2. *Sulphuretted hydrogen.*

This, absent in pure air, is next to CO_2, the most important gaseous impurity in ship air. Its usual sources are human and other animal excretions, and the decomposition of sulphates in salt-water leakage, by rotten timber. Tested

at night by blotting paper charged with Acet. Plumbi, the following table shows :

1st. That SH_2 pervades and to a certain extent surrounds a ship.

2nd. That it is most abundant below, especially on the middle and lower decks, where the men are berthed.

3rd. That the reaction was most decided in the lowest tier, especially the bilge; doubtless not so much from the presence of the SH_2 as of sulphide of ammonium, the chief cause of the familiar bilge smell of wooden ships.*

TABLE 8.—*To contrast the relative abundance of SH_2 in different parts of a ship; temp. 82° F. near Barbadoes; exposure 12 hours (11 p.m. to 11 a.m.); ventilation fair; north-east trade wind.*

	UPPER TIER.						MIDDLE TIER				LOWEST TIER.					
	Upper deck	Half deck.	Main deck.	Main deck cabins.	Cadets' study	Cadets' mess room.	Chest room	Ward room	Steerage.	Lower deck.	Engine room	Stokehole.	Store room	Cells.	Screw alley	Bilge.
A trace	1	1	...	...	.		.	.	. .	...	.	...	...	...		...
Decided trace	...	.	1	1	1	1	1	1	1	1	1	...	. .	. .	...	..
Very decided trace	...	...	...	...	...	...	...	...	...	...	...	1	1	1	.	...
Strong trace	...	...	...	.	..	...	...	...	...	...	...	...	...	...	1	...
Very strong trace	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1

The following results of 25 experiments† at different periods of the night and day show (Table 9) :

* The musty odour which sometimes emanates from the lining of wooden ships doubtless often arises from mould or minute fungi.

† Determined by absorption by water. To a 1½ gallon glass jar filled with the air to be examined, 8 oz. of distilled water was added, the jar then corked

1st. That the actual quantity of free SH_2 varied from ·000578 to·049049 volumes per 1000.

2nd. That it increased with the crowding and the impurity of the bilge.

3rd. That the quantity was greatest when CO_2 was abundant (*e. g.* 16·09 vols. on the lower deck), and the air generally most impure.

TABLE 9.—*Sulphuretted hydrogen.*

	Volumes of SH_2 per 1000.
Lower deck	·000578 to ·049049
Ward room cabins ...	·014714
Bilge	·0490494
Cell (one occupant) ...	·000425

3. *Ammonia.*

The usual sources of this are—human urine, the excreta of fowls, combustion of coal, animal and vegetable decomposition in the bilge, &c. The brownish discoloration of logwood paper showed (Table 10) :

1st. That this highly diffusible gas, or its compounds, is always and everywhere present in ship air.

2nd. That it is most abundant when and where there is most crowding, *e. g.* in the sleeping berths, mess rooms, studies, and occupied cells.

3rd. That it was less abundant in the clean bilge and hold tier generally.

tightly, frequently shaken for 12 hours, and then tested by Sol. Iodid. Potas. and starch, according to the formula given in Parkes's 'Practical Hygiene.' It is much to be regretted that we have no such delicate test for SH_2 as Nessler's beautiful one for ammonia.

TABLE 10.—*Ammonia; relative abundance.*

	Upper tier.						Middle tier.				Lowest tier.						
	Upper deck	Half deck	Cadets' study	Cadets' mess room	Main deck	Main deck cabins	Chest room	Ward room	Steerage	Lower deck.	Store room.	Cells	Engine room	Screw alley	Stokehole.	Cockpit.	Bilge.
A trace	...	...	...						...		...	...	...	...	...	1	1
Decided trace	1	1	...	...	...	...			...	...	...	...		...	...	...	...
Very decided trace	...	...	1	1	1	1	1	1	1	1	1		1	1	1	...	...
Strong trace	...	...	...	...			...		...	...	...	1		...	...	...	...

The following results of 15 experiments by Nessler's test,* at different periods of the twenty-four hours, shows (Table 11):

1st. That its quantity varies from ·0008 to 3·029 average ·31857 grammes per 1000 cubic feet of air, a considerable excess over the trace found in pure air, too small, according to Fownes, for direct estimation.†

2nd. That the highest amount was found on the lower deck at 10 p.m., shortly after the crew had turned in during rough weather and a closed-in ship, when the CO_2 was 21·54 volumes.

3rd. That it was also high on the lower deck during dinner, viz. 0°1699 grammes.

4th. That it is high in confined, badly ventilated cabins.

5th. That in the bilge it rose to 1·088 grammes when this had not been sluiced for some days.

* Determined by absorption by water. To a 1½ gallon glass jar filled with the air to be examined, 8 oz. of distilled water was added, slightly accidulated by Acid. Sulph. Dil., as recommended by H. T. Brown ('Proc. Roy. Soc.,' vol. xviii, p. 287, foot-note). The jar was then corked tightly, frequently shaken for 12 hours, and tested by Nessler's solution in the usual way.

† Roscoe gives the quantity as 1 volume in 1,000,000. According to H. T. Brown ('Proc. Roy. Soc.,' vol. xviii, p. 287) the extreme results on record vary as much as from ·135 to ·0001 parts for 1000 of air.

6th. That in the bilge it probably exists chiefly in the form of sulphide, and elsewhere as carbonate.

TABLE 11.—*Ammonia.*

		Number of experiments made.	Ammonia in grammes per 1000 cubic feet of air		
			Highest.	Lowest.	Average.
Upper tier.—Main deck......		2	0·0001947	0·000138	0·0001663
Middle tier	Lower deck ...	7	3·0299164	0·0003883	0·4845291
	Chest room ...	1	...	...	0·0060859
	Steerage cabin	1	...	...	0·0424784
Lowest tier	Bilge	3	1·0888767	0·000138	0·447961712
	Cells	1	...	...	0·0000859

4. *Ozone.*

Moffat's test paper and Negretti and Zambra's scale (1 to 10) showed (Table 12) that at night, the crew below, when the ozone in the external air was 2, it gradually diminished internally, and was almost wanting in the lowest and inmost parts. This corresponds to its absence in cities (Angus Smith) and hospitals (Parkes); and is to be similarly explained, viz. by its rapid expenditure in oxydating organic matter.

TABLE 12.—*To contrast the ozone in different parts between 11 p.m. and 11 a.m.*

CONDITIONS.		UPPER TIER.					MIDDLE TIER				LOWEST TIER.						
At sea, near Barbadoes; fine light N.E. trade wind, and occasional showers.	Upper deck	Half deck	Cadets' study (empty)	Cadets' mess (empty).	Main deck.	Main deck cabins.	Chest room (full).	Ward room	Steerage (full)	Lower deck (full)	Store room.	Cells.	Engine room	Screw alley.	Stokehole.	Cockpit.	Bilge.
Figure on ozone scale...	2	2	1½	[illegible]	1	1¼	¼	¾	¼	¼	A trace	A trace	A trace	A trace	A trace	A trace	A trace

5.—*Oxydisable organic matter.*

A minute quantity (1 grain in 200,000) of organic matter is found even in the purest mountain air (Watts), also in pure country air, and necessarily more abundantly in town air, its source, in all, being animal and vegetable emanations. It doubtless exists in pure sea air though to a less extent.

The chief sources of this important impurity in ship air are, exhalations from the crew, cargo, and ship, especially the first. Hence, it is most abundant below, and when and where there is most crowding. Hence, also its amount in many cases closely corresponds to, and rises and falls with, the CO_2. Thus, Table 13 shows:

1st. That it takes only from 5 to 12 cubic feet of the air examined to decolorise 1 milligramme of potas. permanganate.*

2nd. That it is abundant in lower-deck air at night.

3rd. That it is very abundant in the confined cells when occupied, and the CO_2 therefore high.

In warm and especially tropical weather the organic matter is greater and more evident to smell, because of the increased exhalations from the crew and the more stagnant and septic state of the air. It is the volatile organic matter, especially that thrown off by the skin, which gives ship air where there is most crowding and least ventilation, as in the cells and lower deck at night, its close and often nauseous smell. Hence why, though perhaps as highly laden with CO_2, the air of officers' cabins, mess rooms, and sleeping berths, has a less sickly odour than that of a lower deck, which possesses few or seldom used baths and lavatories, so essential to cleanliness and health. Nevertheless, as pointed out by Parkes, the quantities of organic matter and CO_2

* The quantity of oxydisable organic matter was determined by slowly passing the air through the permanganate solution by the aspirator described in the 'Lancet,' Dec. 28, 1872. Parkes states that as the quantitative relations of the organic matter of air and potassium permanganate are not yet accurately known, the result should be simply noted, as done in Table 13.

usually bear a certain relation, especially among persons of corresponding cleanliness or carelessness, and increase or decrease together, because the sources of the two impurities are the same, viz. the skin, lungs, and mucous membrane.

TABLE 13.—*To show the quantity of oxydisable organic matter in various parts, and its relation to the* CO_2.*

	Carbonic acid in 1000 volumes of air.	Air required to decolorise ·0001 grammes (= milligramme) of potas. permang.
Cells, 6 p.m. ...	31·823 ...	5 cubic feet.
Lower deck, 10 p.m. ...	8·991 ...	10 „
Do., 3 a.m. ...	14·76 ...	12 „

6. *Watery vapour.*

Watery vapour is usually more abundant in ship than in ordinary air, its chief sources being exhalations from the lungs, skin, ship, &c. In 130 observations in tropical and temperate latitudes it varied from 4·5 to 12 grains per cubic foot, *i. e.* relative humidity 69 to 95, average 79 per cent.; a considerable excess over the 65 to 75 per cent. best fitted for health (Parkes) in temperate latitudes. Table 14, for 3 a.m. the crew berthed (as in Table 1), and the ship housed for the night at Ascension, temp. 78° F. (external air) shows :†

1st. That it is most abundant, exceeding that of the open air, on the crowded lower and main decks, also in the confined ill-ventilated store rooms with seven sleepers, and, worst of all, in the adjacent "cells."

2nd. That elsewhere it diminishes *pari passu* with the crowding, and was least where empty.

* It should be remembered, however, that the unavoidable presence of sulphuretted hydrogen and sulphide of ammonium in small quantities in ship air, is apt to interfere with the accuracy of the results given in this table. It is to be regretted that we have not a more accurate chemical test for organic matter than the permanganate.

† These and the following results were obtained by the ordinary wet and dry bulb-thermometer and Glaisher's tables.

3rd. That it is also lessened by good ventilation and fires, as in the stokehole, the lower deck near the funnel casing, and main deck near the galley, where the humidity was less than in the external air and nearest the healthy standard.

TABLE 14.—*To contrast the humidity in different parts of a ship.*

Place.		Watery vapour. Grains per cubic foot.	Watery vapour. Relative humidity.
External air		6·74	79
Upper tier	Main deck	9·39	94
	„ opposite galley	6·87	71
	Half deck	6·57	77
	Study	6·74	79
Middle tier	Lower deck	10·10	95
	„ opposite funnel ventilator	6·96	79
	Ward room	7·23	82
Lowest tier	Cockpit	7·19	82
	Engine room	7·00	82
	Stokehole	5·70	69
	Bilge	6·24	78
	Store room (7 occupants)	9·69	94
	Cells (1 occupant)	10·43	95

Table 15 shows that the humidity of ship air during the twenty-four hours rises and falls with the crowding, and is greatest at dinner when "all hands" are below (9·6 grains = rel. humid. 80).

TABLE 15.—*Humidity of ship air as affected by crowding; lat. 7° S.; Atlantic; temp. 79° F., external air.*

Place and conditions.		Watery vapour. Grains per cubic foot.	Relative humidity. Saturation = 100.
August 21st	9½ a.m., lower deck, empty	8·603	76
	Noon " full	9·624	80
	5½ p.m. " empty	8·150	72
	7½ " " full	9·356	80
	11½ " " two thirds full	9·056	80

The following corroborates Table 14, and shows that defective ventilation increases the humidity of ship air (Table 16).

TABLE 16.—*Humidity of ship air as affected by ventilation.*

Conditions.			Watery vapour. Grains per cubic foot.	Relative humidity. Saturation = 100.
Lower deck	11 p.m.	Barbadoes, riding head to wind	10·22	85
		At sea, with better ventilation	8·95	68
	3 a.m.	Bermuda, wind nearly aft	10·18	90
		At sea, with better ventilation	7·93	77

The humidity is also increased by washing decks, clothes, bathing, &c.; thus—

TABLE 17.—*Humidity of ship air as affected by washing decks, &c.*

Conditions.		Lower deck.		Open air.	
		Grains per cubic foot.	Relative humidity.	Grains per cubic foot.	Relative humidity.
Washing lower and main decks, temp.	Lower deck, 82° F ..... Open air, 80° F......	9·569	85	8·784	80

The humidity is necessarily greatest under a combination of these causes, *e. g.* when the crew are below and perspiring, as when washing decks, at sea and the ship much shut in, especially in the warm and humid tropics.

On the other hand, steaming diminishes the humidity of ship air by heating it and the ship throughout; thus, Table 18 shows that when the external and relative humidity was 80 it was only 68 on the lower deck, *i. e.* nearer the boiler fires.

TABLE 18.—*Humidity of ship air as affected by steaming.*

		Watery vapour. Grains per cubic foot.	Relative humidity. Saturation = 100.
Steaming in equatorial doldrums, 11 p.m.	Air, external, temp. 82° F.	9·36	80
	„ main deck „ 82° F.	8·86	76
	„ lower „ „ 86° F.	8·95	68

In the stokehole, where the temperature was 107° F., the humidity was doubtless much less. But as Glaisher's tables do not range so high, the exact quantity cannot be calculated.

Ship air is more humid at sea than in harbour, like the

external air. Like this also, the humidity of ship air is greater in tropical than in temperate regions; thus—

TABLE 19.—*Humidity of ship air in temperate and tropical climates.*

Place and conditions.		Watery vapour. Grains per cubic foot of air.	Relative humidity. Saturation = 100.
Lower deck, crew at dinner	Cape of Good Hope, lat. 33° S., temp. 59—69° F.	5½—6½	73—74
	Equatorial doldrums, lat. 5° N., temp. 84—87° F.	10—12	82—87

7. *Suspended matters.*

The fine dust in ship air consists chiefly of *débris* of wood, metal, sails, rope, &c., from the ship, and particles from the bodies, clothing, food, &c., of the crew. Three grains of fine greyish dust from between the beams of the lower deck burnt and smelt like tinder, leaving 0·8 grain of a brownish-black indestructible ash, thus consisting of—

Inorganic matter . . .	26·66 per cent.
Organic „ . . .	73·34 „

while the microscope showed fibres of cotton, linen, wo
epithelium and gritty matter (Plate III, fig. 1). In the
of the lower and main decks passed through an aspir
were seen minute living acari (fig. 2, *A. Farinæ ?*), legs,
of insects (fig. 3), small, round, and oval bodies, diameter
inch (figs. 1 and 3), probably identical with those observ
by Lemaire in the air of a prison and the sweat of a dir
person, and also by Trautman in air, especially when impur
(Parkes). As with ordinary air, important ef results
would doubtless follow a systematic micros

of ship air, especially during the presence of contagious diseases (Class A).

8. *Temperature.*

As regards the temperature of ship air the following table (20) shows—

1st. That, like the external air, ship air generally is cooler by night than by day, the difference being greatest in summer.

2nd. That in winter, owing to fires, crowding, and a closed-in ship, the interior is considerably warmer than the open air, *e. g.* from 7° to 12° F. or more by day, and from 13° to 20° F. or more by night. Again, in summer, while the internal and external temperatures are more equal, the external is warmer by day from solar heat, while by night the internal is from 7° to 10° F. warmer from crowding and closing in.

3rd. That, as a rule, and for an obvious reason, ship air is 1° or 2° F. cooler near the mouth of hatchways, ports, scuttles, windsails, ventilators, &c., opening externally, and correspondingly warmer near those opening from below.

4th. That ship air, both by night and day, is warmest near fires, and especially when the ship is most shut in and where the crew is most crowded.

5th. That it is warmer on the lee than the weather side of the ship.

6th. That it is warmer close to the beams and sleeping crew than near the deck.

7th. That, notwithstanding crowding, closing in, fires, &c., the temperature of a ship's interior may, especially by night and in winter, be cooler than consistent with comfort and perhaps health.

8th. That the above laws hold good for the tropics, although, because of fewer fires and lights, there is less difference between the external and internal temperatures. Again, by day the greater stagnation and humidity of ship air, and slower cutaneous evaporation, make it, though actually lower in temperature, *apparently* warmer than the external air,

while by night the same causes *apparently* increase its greater heat.

TABLE 20.—*Actual and relative temperatures of ship air.*

Place.	Temperate zone. H.M.S. "Britannia."				Tropics. H.M.S. Bristol.
	Summer, May 27th, 1872.		Winter, October 24th, 1873.		October 6th, 1871.
	Day, 1 p.m.	Night, 10 p.m.	Day, 11½ a.m.	Night, 11½ p.m.	Night, 10 p.m.
External air (shade)	68	51½	38½	30 and under	77
Main deck.—Studies	63½	61½	48	49	...
Weather side	66	60	47½	49½	78½
Lee side	...	...	...	...	79½
Near fore hatch, leading below	...	...	...	...	80
Near a starboard port	...	...	...	...	78
Middle deck	65	56	46	50	...
Lower deck.—Weather side	64	55½	51	43½	81
Lee side	...	...	...	...	82½
Near beams	...	...	...	...	82½
Near deck	...	...	...	...	82
Near mouth of a windsail	...	...	...	...	78½
Near fore hatch, leading on deck	...	...	...	...	80½
Near main hatch, leading on deck	...	...	...	...	79½
Near funnel, casing ventilator	...	...	...	...	80
Officers' cabin, weather side	...	...	...	...	79½
Officers' cabin, lee side	...	...	...	...	[illegible]
Cockpit	62½	54½	48	47½	[illegible]
Model room (after cockpit)	61½	54	47	46	[illegible]
Engine room	...	...	...	...	[illegible]
Hold	59½	...	45½	45	[illegible]
Stokehole	...	...	...	...	79
Bilge	...	...	...	...	79
Highest	66	61½	51	50	82½
Lowest	59½	54	45½	43½	78
Range	6½	7½	[illegible]	[illegible]	4½

The following analysis of bilge water, which so often contaminates ship air, will be interesting. One litre, collected seven days after the bilge had been sluiced, was of sp. gr. 1031 and temp. 74° F. (sea water being sp. gr. 1027 and 71° F.). Supernatant fluid tolerably clear, strongly alkaline, smell fetid, test paper charged with Acet. Plumbi becoming dark by five minutes' close exposure to its surface.* Sediment copious, dark, of a highly offensive odour, and showing under the microscope small particles of wood, sand, coal, whitewash, &c., its weight being—

4·082 grammes { 2·527 grammes inorganic matter, effervescent and partly soluble in acid. sulphuric. dil.
1·555 grammes organic matter, destructible by heat. }

The basis of bilge water is sea water, with certain added soluble and insoluble foreign matters. Of the soluble, 1 litre contained—

1. Sulphuretted hydrogen (test, Sol. Iodin. and starch) =0·059925 gramme or 2·54 cubic inches = 0·0564 gramme of sulphur.

2. Ammonia (test, Nessler's) = 0·1269 gramme, probably chiefly sulphide.

3. Sulphides (test, Sol. Acet. Plumbi in soda) abundant, especially sulphide of ammonium, but the quantity was not determined.

4. Lime in solution (test, Oxal. Ammon.), 2·268 grammes of oxalate of lime = 0·697 gramme of lime (Ca 2HO).

5. Carbonic acid (test, Aq. Calcis), a slight deposit.

6. Organic matter; 1 litre decomposed 0·141 gramme of permanganate of potas. almost instantaneously. However, free SH_2 and sulphide of ammonium necessarily render this test and result to some extent doubtful.

* This was a comparatively mild specimen of bilge water.

9. *Medical bearings of the case.*

The chemical interest attached to the present inquiry is surpassed by its medical importance, inasmuch as it leads to the threshold of another still more abstruse, viz. the sole or joint effect of impure ship air on the physique of seamen, and on the origin, character, and course of the various diseases prevalent among them. As a rule, our most successful preventive and curative efforts are those based on exact physiological and pathological knowledge. Of the subject here indicated, however, only the leading features can be now discussed, with a few illustrative examples selected from a doubtless fertile field of research.

Seamen, as a body, are neither healthy nor long lived, but the reverse. This is proved, first, by their low average age, said to be 33, evidently too brief for men who are carefully selected, and subsequently kept under the most approved hygienic influences; and, second, by their sick and death rates equalling those of landsmen, who include the two periods which contribute most to both lists, viz. infancy and old age. This indicates subjection to certain unhealthy agencies, of which impure air is evidently one, and perhaps the most powerful. So great is the vitiation that we cannot but wonder that seamen keep their health, such as it is, so well, and can only explain it by their longer subjection on deck to the purest of all air, which, to a certain extent, counteracts the evil effects of the impure air breathed below, and prevents the system from sooner succumbing to its influence. Although ship air may be, and often is, long breathed with apparent immunity, it is really slowly but surely undermining the constitution, partly by poisoning the blood and tissues, and partly by depriving them of oxygen. To its prolonged effect is doubtless partly due the pale sickly hue of men kept much below, and mainly the stunted growth of seamen generally, their premature old age, comparatively early death, and much of the disease which prevails among them.

The more immediate influence of the constant or frequent inhalation of this impure air in the production of disease is effected in two ways, either locally and mechanically, or generally and chemically, thereby inducing many slight and serious, obscure and obvious results. The blood, laden with retained and perhaps reabsorbed carbonic acid, lingers in the lungs, craving to be oxygenated, thus gorging them. Nightly or oftener renewed, this passive congestion becomes progressively greater and more permanent, and the lungs thereby increasingly prone to inflammation. What more likely to excite the latter than the chill resulting from the sudden transition from a warm hammock into the cold external air, and the consequent inward revulsion of blood, especially into the breathing organs, to which the majority of every crew is subjected once, and often twice, every night when roused to keep watch. If then sent "aloft," and both lung and heart function thereby greatly and suddenly increased, the lung circulation is likely to become still further impeded, and, as a sequel, the heart and great vessels secondarily distended, embarrassed, and laboured in action. What is the effect of a frequent repetition of this on the pulmonary and cardiac systems? How many of the 6796 cases of catarrh and other inflammatory lung diseases, and of the 392 cases of morbus cordis, which occur annually among the 50,347 originally strong, healthy, selected men in H. M.'s Navy, are to be thus directly or indirectly accounted for?*

Without attempting to specially consider the individual effects of organic matter, sulphuretted hydrogen, and other impurities, beyond noticing their undoubted and even, according to some, paramount unhealthy influence,† it will be

* These averages are for three years (1867–8–9) taken at random.

† The most important class of diseases produced by impure air is certainly caused by organic matters, especially floating ones (Parkes). The respiration of an atmosphere only slightly impregnated with sulphuretted hydrogen may, if long continued, seriously affect an individual, and even cause death (Taylor). Ammonium sulphide is still more poisonous; but authorities are much divided regarding the quantities which injuriously affect health, doubtless because their influence, like that of other poisons, varies according to

obvious that as the blood gets laden with these, and especially carbonic acid, the function of the red corpuscles as oxygen carriers, and that of the white globules as fibrin elaborators, becomes impaired, and the liquor sanguinis thus soon chemically and vitally altered. Instead of oxygen it carries poison, and hence, instead of healthily stimulating, it narcotises the nervous, muscular, glandular, and other tissues and systems, producing certain effects, immediate and remote. Hence, for example, the lethargy, headaches, nausea, malaise, and other minor ailments common on shipboard, especially among the newly called night-watch, and officers in crowded studies, cabins, and mess rooms, especially in the tropics. Moreover, impure blood yields faulty gastric, hepatic, and other secretions, and thus vitiates digestion and primary assimilation. While by the resulting limited production of albumen, aided by the impaired function of the white blood-corpuscles and the deficient supply of oxygen in ship air, secondary assimilation and nutrition, including growth and the ordinary renewal of tissue, become defective.

The doubly imperfect nutrition resulting from vitiated air and faulty dieting is exactly the condition in which struma is most likely to be acquired, or developed in an hereditarily predisposed subject; hence, doubtless, the 384 cases of scrofula which occur annually in H. M.'s Navy; and the above-noted mechanical obstruction explains why, in seven eighths of these, its semiorganised deposit is determined to the gorged lungs, to manifest itself in the imperfectly nourished lung-tissue in the form of phthisis. Baudelocque's belief that vitiated air is the real source of the strumous diathesis, and that other agencies usually accredited with the production of phthisis, *e. g.* deficient exercise, want of cleanliness, exposure, &c., though important aids, are only secondary and subordinate causes, of themselves non-effective, is doubtless correct, as are the

individual susceptibility, and the state of health at the time of subjection to them. One thing, however, is certain, they are all abnormal and unhealthy, and doubtless add to the general result.

opinions of the Army Sanitary Commissioners, Bryson, Milroy, and others, who ascribe the great prevalence of phthisis in the army and navy chiefly to impure barrack and ship air. Blood vitiated and tissues impoverished to this extent fall an easy prey to disease on the addition of a fresh blood poison, whatever its nature; and this explains why the semi-scorbutic, rheumatic, and other blood diseases are predisposed to, and more prevalent among seamen than they might be.

Whatever the true nature of contagious diseases, especially the exanthemata, impure ship air unquestionably aids their propagation, both by affording their germs a suitable medium in which to become developed, and predisposing the blood and system to receive and multiply them. Erysipelas (which led to the present inquiry), first developed in the heat and humidity of the Equator, continued to occur in H.M.S. Bristol, in accordance with a law already well known, whenever she was more shut in and less ventilated than usual, and her atmosphere thus unusually vitiated, as at Simon's Bay, Barbadoes, the Equator, Bermuda, and near the Azores (see tables), where the CO_2 varied from fourteen to twenty-four volumes per 1000, organic matter and other impurities being correspondingly increased. Fig. 4—blood from an erysipelatous leg, in which a minute active animal was found about one third the diameter of a red blood-corpuscle * (diameter $\frac{1}{12000}$ inch), is given more as suggestive of continued examination of the blood in contagious diseases than as offering any explanation of the nature of erysipelas. However, whether these organisms were the cause or merely the result of this disease, I have more recently (1872) seen a similar equally active animalcule in the blood of an erysipelatous scalp.

The high impurity of modern ship air suggests how much greater must have been the vitiation of former times in smaller, more crowded, and less carefully ventilated vessels. Nor are their high sick and death rates surprising, or the great

* Seen also by my colleague, Surgeon Charles Fennel, R.N.

prevalence of scurvy, putrid fever, sloughing ulcer, dysentery, and other diseases arising from blood poisoning. The same cause is still at work among seamen, though in a minor form, producing similar widespread, though necessarily less marked, pathological effects. All medical authorities agree regarding the highly injurious effects of the various impurities found in ship air; and remembering that pure air is one and the most urgent of the indispensables of health, and hence that an atmosphere, at once so vitiated and so constantly undergoing recontamination as ship air, cannot be too often renewed, the facts here adduced suggest various sufficiently obvious hygienic indications, not only regarding ships of war, but vessels of every nation, kind, and class. We cannot on shipboard, as on shore, except to a trivial extent, reduce the vitiation by increasing the cubic air space, and are thus compelled to adopt the only other available plan, viz. more frequently renewing the air, *i. e.* increasing the ventilation; and, regarding this, it must be obvious—1st, and more especially, that from the beginning to the end of a ship's active career her ventilation should not be deemed of secondary but of primary importance; 2nd, that ships should have the best possible ventilating apparatus; 3rd, that this should be always efficient, ever at work, and constantly looked after, even if "automatic" or "self-acting;" 4th, that increased attention to ventilation is necessary at certain periods, especially at night, during meals, and peculiar conditions as to crowding, direction of wind, &c.; and 5th, that, so necessary is a constant and complete renewal of air to attain the greatest possible purity, we should not trust solely to any single system, but in turn or conjointly employ propulsion, suction, and diffusion, aided, whenever possible, by other means, as scattering the men towards the best ventilated parts, &c. The more we perfect ship ventilation and the purer we make the atmosphere in which seamen live, the healthier will navies become. There is no apparent reason why ship air should not be as pure, if not purer, than barrack or house air. To make seamen thoroughly healthy the unsanitary agencies to which they are subjected must be

altogether, or at least in great measure, removed; and if we succeed in removing this, doubtless the most powerful of them all, they will as a class become healthier and longer lived than landsmen, and even than soldiers.

DESCRIPTION OF PLATE III.

FIG. 1.—Fine dust from lower deck. (Magnified 30 diameters.)

FIG. 2.—Living acari from lower deck, night air. (Magnified 30 diameters.)

FIG. 3.—Small round and oval bodies in lower deck, night air. (Magnified 30 diameters.)

FIG. 4.—Blood from an erysipelatous leg containing animalcules (*a*, magnified 250 diameters); ditto (*b*, magnified 500 diameters).

Fig. 1
Mag. 30 diam.

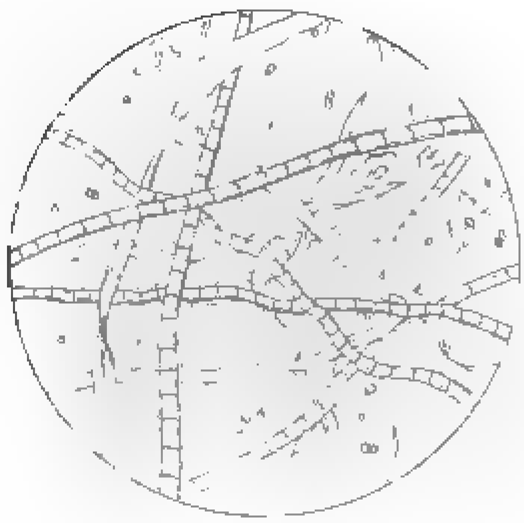


Fig. 2
Mag. 30 diam.

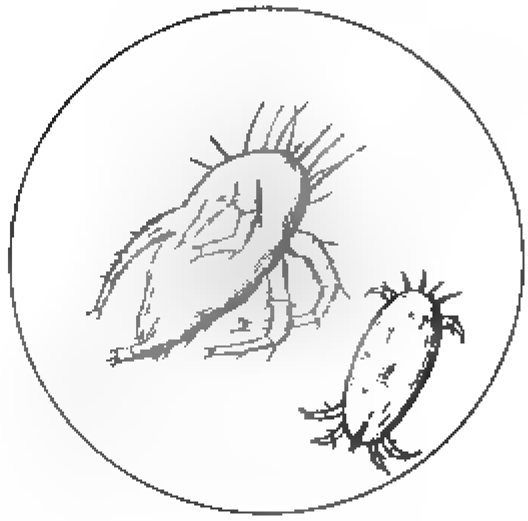


Fig. 3
Mag. 30 diam.

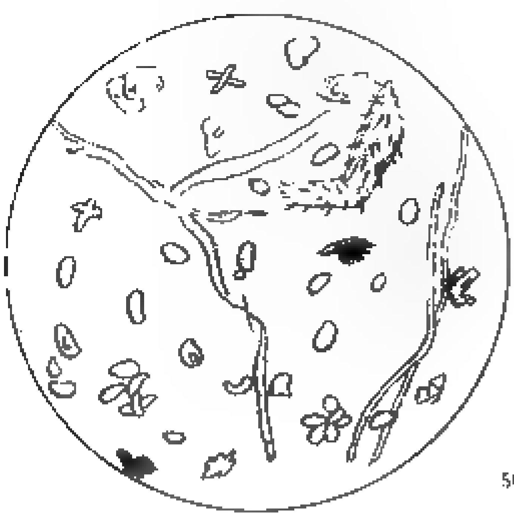
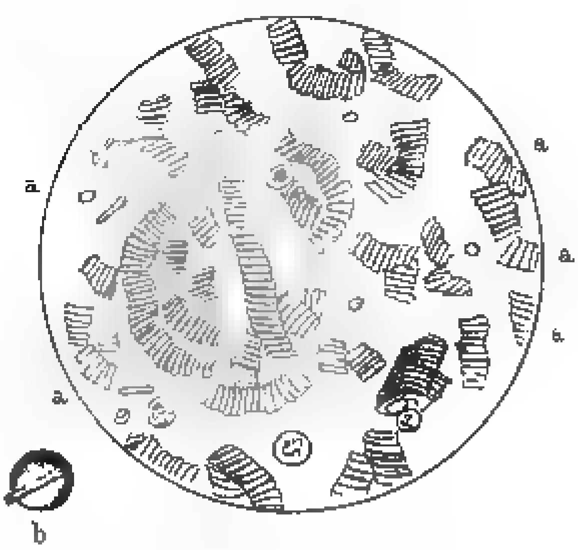


Fig. 4.
Mag. 250 diam.



G. H. Ford

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A SECOND REPORT

ON THE

COMMUNICATION OF SYPHILIS IN THE PRACTICE OF VACCINATION.

WITH TWO ADDITIONAL CASES.

BY

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Received January 16th—Read January 28th, 1873.

In the spring of 1871 it became my duty to ask the attention of the profession, in the most prominent manner that I could, through the medium of this Society, to two series of cases in which syphilis had been communicated to numerous individuals in the practice of vaccination. There was nothing new to medical science in the nature of the facts, for closely similar ones had occurred previously in various parts of the Continent, and had been faithfully recorded. The narratives of them had also excited much attention amongst ourselves, and had been criticised and investigated by many writers, amongst the ablest of whom I must mention the name of Dr. Ballard, who in his work on Vaccination had treated the subject exhaustively, had avowed his conviction of the reality of the occurrence, and had anticipated almost all the observations which have

occurred in connection with subsequent cases. Nor had we in English practice been wholly free from such accidents, for, not to mention some more or less suspicious occurrences which were not placed beyond doubt, a case which was perfectly clear in all its details had been brought by Mr. Thomas Smith before the Clinical Society six months prior to my own paper. Notwithstanding these circumstances, however, I believe that the facts which I brought before the Society produced in the British profession a sense of most disagreeable surprise, and awoke many for the first time to the perception of a danger which they had never before realised, and of which most had scarcely credited the existence. In the minds of some, I believe that I incurred blame, as being unwise and rash in making such facts public. I could not myself think so at the time, nor do I now, for it seemed most necessary that the knowledge of such a danger should be diffused as promptly and widely as possible. This opinion has been confirmed by the circumstance that during the last eighteen months two other examples of the communication of syphilis in vaccination have come under my notice. One of these presented itself amongst my patients at Moorfields, where the man applied on account of iritis and without the slightest idea as to what he was suffering from, and the other was brought to my house as a private patient, the lady being the subject of a urethral growth. I wish particularly to state that neither of the cases was sent to me because I had taken an interest in vaccination-syphilis, but that both showed themselves in the ordinary course of practice. It may be recollected that neither of my first series came under observation in this way, but that in both the nature of the disease had been recognised by others before I was consulted. I purpose in the present paper to relate briefly the particulars of the two cases to which I refer, and neither of which has as yet in any way been made public, and after doing this I shall ask to be allowed to make a few general remarks on the whole of the facts which I have had under observation. I may just add that during the last two years several cases have been sent

to me as supposed examples of vaccination-syphilis, but in none could the diagnosis be supported.

Third Series (one case).

The following case came under my care at Moorfields, where its subject applied on account of acute iritis, of the cause of which he had no suspicion.

The patient was a tobacconist, æt. 46, a married and respectable man. The iritis for which he applied at Moorfields was double. On investigation it was found that his body and limbs were covered with a papular and scaly rash of a very definitely syphilitic nature; he had also symmetrical sores in the tonsils. The examination further revealed the fact that he had large unhealed sores on his arm which had resulted from vaccination. He told us that he had been vaccinated three months before, being at the time in perfect health. Several of his children (young adults) were vaccinated at the same time and from the same child.

His account was that the vaccination places took and went on favorably, but that just as they were about healed, during the fourth week, they again inflamed, became ulcerated, and gave him much trouble. He attributed this relapse of inflammation to the irritation of tobacco dust, in which he worked. It was not until more than six weeks after the vaccination that the eruption showed itself, and not till the end of another month still that the iritis occurred. He had not as yet had any specific treatment, since the nature of his disease had not been recognised. On being questioned as to the possibility of his having contracted syphilis he firmly denied it. He readily submitted to an examination of his genitals, and we could not find the slightest trace of sore on them. His vaccination sores, at the time he came under my notice, were open ulcers as large as shillings, covered with scab and with dusky indurated borders. There was an indolent swelling in his armpit.

This man remained under my treatment at Moorfields

for two or three months, and was seen repeatedly by many members of the profession who were attending my clinique. There could not be the slightest doubt that he was suffering from secondary syphilis, and that too in a very severe form. We treated him with mercury, and both his iritis and rash disappeared.

Everything in the man's history that we could ascertain seemed to point to vaccination as the source of contamination. His vaccination spots were the seat of chancrous induration after having in the first instance healed. The date of their induration and the date of appearance of the rash fitted exactly with the hypothesis that he had in some way acquired syphilis about the date of his vaccination.

Having obtained from him the name of his vaccinator I called on that gentleman, and from him obtained information which strongly confirmed the man's statements. He said that he had never in his life seen such vaccination sores as this man's arm displayed. "He had been quite frightened at them," and had thought their condition so near phagedæna that on two occasions he had applied a strong solution of nitric acid. He had never, however, suspected the real nature of the disease.

About a dozen other persons had, I was informed, been vaccinated at the same time and from the same child as our patient. The vaccinator told me that he knew them all, and that, with the exception of a little trouble in the healing of the sores in one or two, none of them had shown anything peculiar. He was very anxious that we should not excite alarm in the neighbourhood by instituting any inquiry about these patients, and as he promised to get to know quietly how they were doing and to inform me if he found anything suspicious, I thought it best not to press the matter further. I ascertained from our patient himself that his own children, three in number, who had formed part of the series, had all done perfectly well. The vaccinator told me that the infant from whom he had vaccinated was a model of vigour and health, and he courteously procured for me an opportunity of seeing it.

The Vaccinifer.—I found it a very large and very fat baby, eight months old. It had no rash nor any trace of condyloma, and the only point about it suggestive of syphilis was the state of the bridge of the nose, which was decidedly broad and sunken. Its mother appeared in good health, but she told me that her first two children had died in infancy and that this, her third, was the only one she had living.

I have, in my own mind, no doubt from the appearance of the child's nose that it is the subject of inherited taint, and shall confidently expect that future years will prove the correctness of this suggestion; nor do I see any other way in which the symptoms displayed by the man can be accounted for, excepting on the supposition that he contracted syphilis from vaccination. If we suppose that from some other source he acquired a chancre at or near the date of his vaccination, it is remarkable that no trace of such sore should have been left; and it is yet more remarkable that the vaccination spots should have become indurated and have caused an axillary bubo. Nor does it seem in the least probable that the vaccination sores had become accidentally contaminated. No plausible source of such contamination can be suggested, while, as already stated, the dates fit exactly with the supposed introduction of the virus at the time of vaccination. Thus, we seem to have an instance in which only one patient out of twelve vaccinated from the same subject became contaminated; at any rate there is, as yet, no proof that any of the others have suffered, but under the peculiar circumstances of the case I can by no means accept this absence of evidence as conclusive. It is not improbable that something more may in the future be heard of some of these patients.

I will now relate the particulars of the fourth example of vaccination-syphilis which has come under my notice. They are in most points very similar to those of the preceding case; out of three vaccinated only a single individual became diseased.

Fourth Series (one case).

Mrs. M—, aged about 46, came under my care last December. Her ailment was a vascular growth in the urethra, but in the course of the examination I found that she was covered with the stains of a syphilic rash. On my asking about it she told me that she had been very ill after vaccination and had suffered from a severe eruption and from inflammation in one eye.

The following, on inquiry, proved to be the facts of her case:—She was vaccinated in May, 1871, and her two daughters, one aged 30 and another 15, were done at the same time. Four punctures were made in her arm and all of them soon healed, not having taken; but a month later one of them again became sore, and a hard-edged very troublesome ulcer resulted which lasted for three months. Within a few weeks of the formation of this ulcer, *i. e.* within seven or eight weeks of the date of the vaccination, she became covered with rash. It was especially copious on the chest and back of the neck and affected also the palms of her hands. From the date of the vaccination in May until the beginning of September she remained under the care of her own medical attendant by whom the vaccination had been performed. In September, as the rash remained out, she consulted another practitioner. Under her second adviser she remained for nine months taking almost continuously iodide of potassium and bichloride of mercury in small doses. In July, 1872, when the rash had been out eleven months and was still but half cured, iritis occurred in the left eye. It was so severe as to threaten the destruction of the organ, and after some delay she got admitted into the Ophthalmic Hospital. It should be stated that for two months before the outbreak of the iritis she had suspended all treatment and had been staying at the sea-side for the benefit of her health. She was in Moorfields from July 19th to August 1st and was treated by mercury and atropine. The diagnosis given, as I ascer-

tained from her prescription paper, was "Iritis, specific (?), after vaccination (?)" At the time of the iritis her rash relapsed and her palms became covered with scaly patches, her finger-nails also thickened.

At the time that this patient came under my observation she was much out of health and was still covered by the stains of an eruption about the nature of which there could not be the slightest doubt. There were adhesions of the iris in the left eye. I found that she had been much annoyed with the gentleman who had vaccinated her, attributing what had followed to his having done it when she was not in a proper state of health. She had not, however, the slightest idea of the nature of the disease from which she had suffered. Her surgeon had not suspected it either, and consequently, during the first four months, no specific treatment had been adopted. On asking her as to the particulars of the vaccination she told me that it was done from arm to arm in the surgeon's own house. The infant, according to her statement, was puny and its mother looked ill. She was not aware whether the child's arm bled or not. In her two daughters who, with her, were vaccinated from the same child, no definite ill results followed; in both, several spots took, went through their stages and healed soundly at the usual time. In neither of them did any of the spots re-ulcerate, but the younger one twice suffered from transitory rash, probably urticaria. I afterwards called on the surgeon who had vaccinated this patient: he confirmed her statements in all particulars excepting that he did not allow that the vaccinifer looked puny.*

The Vaccinifer.—I succeeded in tracing the vaccinifer. Its mother was the wife of a lighterman and had borne three children previously. Of these the eldest is a boy of ten who now shows no signs of syphilis and who is said to have had no symptoms in infancy. The second was stillborn. The third was a girl of four who had no special symptoms in

* It seems probable that the woman who is the subject of our narrative was the last or last but one vaccinated out of a considerable batch.

infancy excepting that for several months during teething she had very troublesome ulcers at the anus (condylomata). For these she was long under medical care and had numerous local applications. At the same period she was considered to have "a tendency to water on the head." The vaccinifer was four months old at the date of vaccination and appeared, according to his mother's statement, to be perfectly well. Subsequently, however, when dentition commenced he, like his elder sister, had very troublesome sores at the anus and a tendency to water on the head. For the anus he was three months under treatment at a dispensary and had blue stone applied. He is living, full grown, and shows no peculiarities excepting a large forehead.*

There cannot be the slightest doubt in this case that the patient has suffered from syphilis, the only question to be raised is how was it obtained? On this point I hold that a vaccination puncture having reopened a month after healing, having formed a large hard-edged ulcer which lasted three months and which was promptly followed by the rash, is an almost conclusive piece of evidence. The dates of each occurrence are precisely what we should have expected.

The patient is a married woman past middle life, and there is not the least reason for suspecting contagion from any other source. I may add that the vaccination ulcer has left a large dusky scar exactly such as I have seen in other cases of vaccination-syphilis.

Before leaving this case I may ask attention to the fact that the secondary symptoms were unusually severe and protracted, a circumstance not improbably due to the fact

* Since this paper was written I have obtained from the mother the name and address of a surgeon who treated her child. On inquiry he tells me that the child had *syphilitic condylomata*, and was, on and off, under his care for months. He states that it had also snuffles and a slight skin rash, and that it was cachectic and puny. He did not ask the parents any direct questions because he was perfectly certain as to the nature of the ailment.

that no specific measures were employed during the first four months, and that they were subsequently given very insufficiently.

I will now, with the permission of the Society, make a few general remarks on the subject of vaccination-syphilis, chiefly in connection with the four series of facts which I have recorded. It will be convenient to arrange what I have to say under separate headings.

1st. What are we to infer from the circumstance that when syphilis is conveyed in the practice of vaccination it does not affect all of those vaccinated from the tainted source? Clearly, I think, we must believe that the specific poison of syphilis is either not contained in the vaccine lymph at all, or is not equally diffused through it. In my first series of cases two patients out of twelve were successfully vaccinated and wholly escaped syphilis; in the second series out of about twenty-six more than half escaped; and in the third only one out of twelve is known to have suffered, whilst in the fourth only one suffered and six or eight probably escaped.

In the first and second series it was repeatedly observed that of those who contracted syphilis, some of the vaccination punctures developed chancres and others did not. There cannot be the slightest doubt that it is quite easy to vaccinate from a tainted vaccinifer without conveying syphilis, and on the other hand that it is possible to convey syphilis either with or without the production of a normal vaccine vesicle. Now, the supposition that it is necessary to convey some of the cell-elements of the blood in order to convey syphilis, seems to my mind by much the most probable explanation. Probably it is not necessary that these elements should be visibly red. That the vaccine virus itself in a pure state cannot produce syphilis seems highly probable, since in several recorded instances vaccination has been inadvertently performed on a considerable scale from a child that was subsequently found to be syphilitic,

and without ill consequences. It is probable that in a great number of instances, in addition to those placed on record, this has happened, and the evidence supplied by it in reference to the impotency of pure vaccine lymph in the production of syphilis is very strong. On the other hand experiment has fully proved, and more especially the well-recorded experiment of Professor Pelpizzari, that the blood of a patient in the secondary stage of syphilis can, when inoculated, produce a chancre which will be followed by the usual role of syphilitic phenomena. The facts in the case referred to afford as regards dates, &c., a very exact parallel with what was observed in all the cases which I have recorded.

Next we may ask is it absolutely necessary that *blood* should be used in vaccination in order to convey syphilis? It seems highly probable that it is not. At any rate there is not the least evidence in three out of the four series of cases which I have recorded that the lymph used was visibly contaminated with blood. The vaccinator in each instance asserts that it is his habit most scrupulously to avoid making the vesicle bleed. Probably it is quite sufficient to allow the vesicle to draw or weep. With this drainage no doubt corpuscular elements of the blood and tissues become free. According to this supposition as soon as the first contents of the vesicle are exhausted the risk begins. It is well-known that it is the custom of many experienced vaccinators to allow the ruptured vesicle "to weep," and to continue to employ its secretion long after the exhaustion of its original elements.

If the syphilitic virus and the vaccine virus be implanted at one and the same time, what will be the course of events? The cases recorded show conclusively that, if the patient be susceptible to vaccination, the vesicle may pass through all its stages in the most characteristic manner. Then after healing of the vaccination-sore, and at the end of about a month from the inoculation, the syphilitic virus begins to show its effects and the scar becomes irritable, inflames, and indurates. Although this course is the usual one it is not

invariable and deviations from it may be observed in connexion probably with the patient's age, state of health, and condition of tissues.

In these exceptional cases the vaccination-sore never heals, and the pus-scab which forms over it combines with the inflammatory swelling around to conceal the nature of the specific changes which subsequently occur. Should the vaccination not have taken, it is usual for the puncture to heal and for the patient to think no more about it until induration occurs at the end of the month.

What are the usual characters of the vaccination-chancre? As already hinted above, the amount of inflammatory effusion on the surface of the sore, and of inflammatory œdema at its base, may in certain cases be considerable. In several of the cases in my second series the specific characters of the chancre were in this way quite concealed. In these instances the patients were children. In the man who is the subject of my third observation the history was that the sore had been very acutely inflamed, so much so that the surgeon several times cauterised it, and probably it was on the verge of phagedæna. These conditions are, however, exceptional and in a usual way the vaccination-chancre shows but little tendency to excess of inflammation. It some cases it does not even ulcerate. It begins as a little red, firm, glossy tubercle which gradually increases in size and becomes harder. At the end of a fortnight, or earlier, it usually ulcerates and presents a sore remarkable for its small amount of secretion and for the hardness of its base and edges. The cases in which no mercury was given show that it may last for some months before it heals. After healing it leaves a dusky brown scar very different indeed from that of vaccination.

What treatment ought the vaccination-chancre to receive? I can feel no doubt that should a vaccination-scar take on the induration characteristic of a chancre, and should the other facts of the case corroborate the suspicion, it is the surgeon's duty without delay to begin the administration of mercury. The cases which I have recorded show in the

strongest possible light the great difference in result between those in which mercury was given and those to which nothing was done. In my *first series* of cases the nature of the accident was recognised during the sixth week after vaccination and prior to the occurrence of any well-marked secondary symptoms. In all the patients, excepting one, mercury was at once commenced and in all these the progress of the chancre was at once arrested and rapid cure resulted. For a considerable period no secondary symptoms shewed themselves, and the success of the treatment was such as to induce not a few to doubt the correctness of the diagnosis. Subsequently, however, secondary symptoms showed themselves in several of the patients. They were so well characterised as to put all scepticism about the nature of the disease out of question, but still they were comparatively very slight. They yielded very quickly to the renewed administration of mercury and none of the patients in any material degree lost health either from the disease or the remedy, iritis did not occur in any single one and I believe they are all at the present date quite well. The only case which gave any real trouble was that of a young woman in whom suppuration in the cervical glands took place, and in her most probably it was strumous rather than syphilitic. The contrast in this respect was very great between the *first* and *second series*, and still greater in respect to the two cases which I record in the present paper (*third* and *fourth* series). In neither of the two latter was the nature of the disease suspected until the skin was covered with secondary rash. In both the chancre on the arm became very large and remained open for several months. In both the eruption came out most copiously and was attended by great loss of flesh and strength. In both iritis of a very severe character occurred. One of them was cured, both as regards local phenomena and general health, by a course of mercury; in the other the disease under inefficient treatment has lingered for twenty months and the patient is still suffering much from its effects. It is of course too

early to obtain data as to the relative liability of the patients to the tertiary forms of syphilis, but so far as the primary and secondary symptoms are concerned I cannot speak too strongly as to the vast apparent advantage of the mercurial plan. The lesson of the cases is very clearly opposed to the too prevalent modern doctrine that it is well to wait for secondary symptoms before beginning specific treatment, and would appear to indicate that the latter should be adopted as soon as ever the condition of the chancre permits of an accurate diagnosis. I may also in passing be permitted to ask attention to the interesting illustration which these cases afford of the manner in which mercury interrupts the evolution of syphilis and delays the occurrence of secondary symptoms. In all the cases which were not treated secondary symptoms showed themselves from the sixth to the ninth week after the inoculation, whilst those treated by mercury did not show symptoms until from five to seven months afterwards.

In conclusion a few words must be said as to the best means by which we may hope to prevent the occurrence of these lamentable accidents in future. Foremost under this head I would put the diffusion of the knowledge amongst the profession that such accidents are possible. Until quite lately almost the whole British profession was incredulous on this point, and in spite of the publicity which was given to the facts two years ago there still remain, I believe, many who are either uninformed or unconvinced. The vaccinator who proceeds in his duties with the fear of syphilis before him can I think incur but little risk in the matter. He will in the first place select his [illegible] carefully, avoiding all children whose parents are [illegible] to him. He will for the most part avoid all [illegible]

* Of the vaccinifers in my four series of cases two were first-[illegible] the third was the first which had lived, the parents having lost [illegible] children in early infancy, while in the remaining case, although [illegible] a brother and sister living, and apparently in good health, there [illegible] that the latter had in infancy long suffered from [illegible] at the [illegible] bound, however, to admit that if this the sec[illegible]

children and wait until by the development of one healthy child some guarantee of freedom from taint on the part of the parents has been given. There certainly cannot be any difficulty under ordinary circumstances in procuring vaccinifers who are absolutely free from risk. Next to the scrupulous selection of the child from whom to vaccinate come the obvious precautions of avoiding the use of blood and of recent exudation from the walls of the vesicle, but these and many other matters of detail have already been so well enforced that it is needless to allude to them further.

vaccinifer, there would, as far as can be ascertained by inquiry now, have been no facts likely to arouse the suspicion of the surgeon. The mother would have been able to produce the guarantee of an older child in good health.

REMOVAL OF A NEEDLE FROM THE HEART.

RECOVERY OF THE PATIENT.

BY

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SURGEON TO ST. BARTHOLOMEW'S HOSPITAL.

(Received January 14th—Read February 11th, 1873.)

THE following case is, I believe, the only instance on record in which a patient has recovered after the removal by a surgical operation of a needle from the heart.

J. E—, æt. 31, a pewterer, during a struggle in a public house missed a needle which he had placed in the left side of his coat, and he fancied that it must have entered his chest. The day after, feeling some pain in the left side, he came to St. Bartholomew's for advice. No wound was to be seen, either then or later, and as nothing could be felt on examining the chest no treatment was at this time indicated. For nine days he followed his ordinary occupation, but all these days he was in pain, until at length the discomfort became so great that he again applied for relief.

On October the 28th, 1872, he was brought under my notice in the consultation room. He was a pale, but fairly well-nourished man, in good general health and with a good family history. He told me he had been troubled since the

occurrence of the presumed accident with what he called "beating" of the heart, and had pain, which he very consistently described as continuous, extending from the left nipple towards the corresponding armpit, and thence down the inner side of the arm to the elbow, apparently along the course and in the distribution of the lesser internal cutaneous nerve.

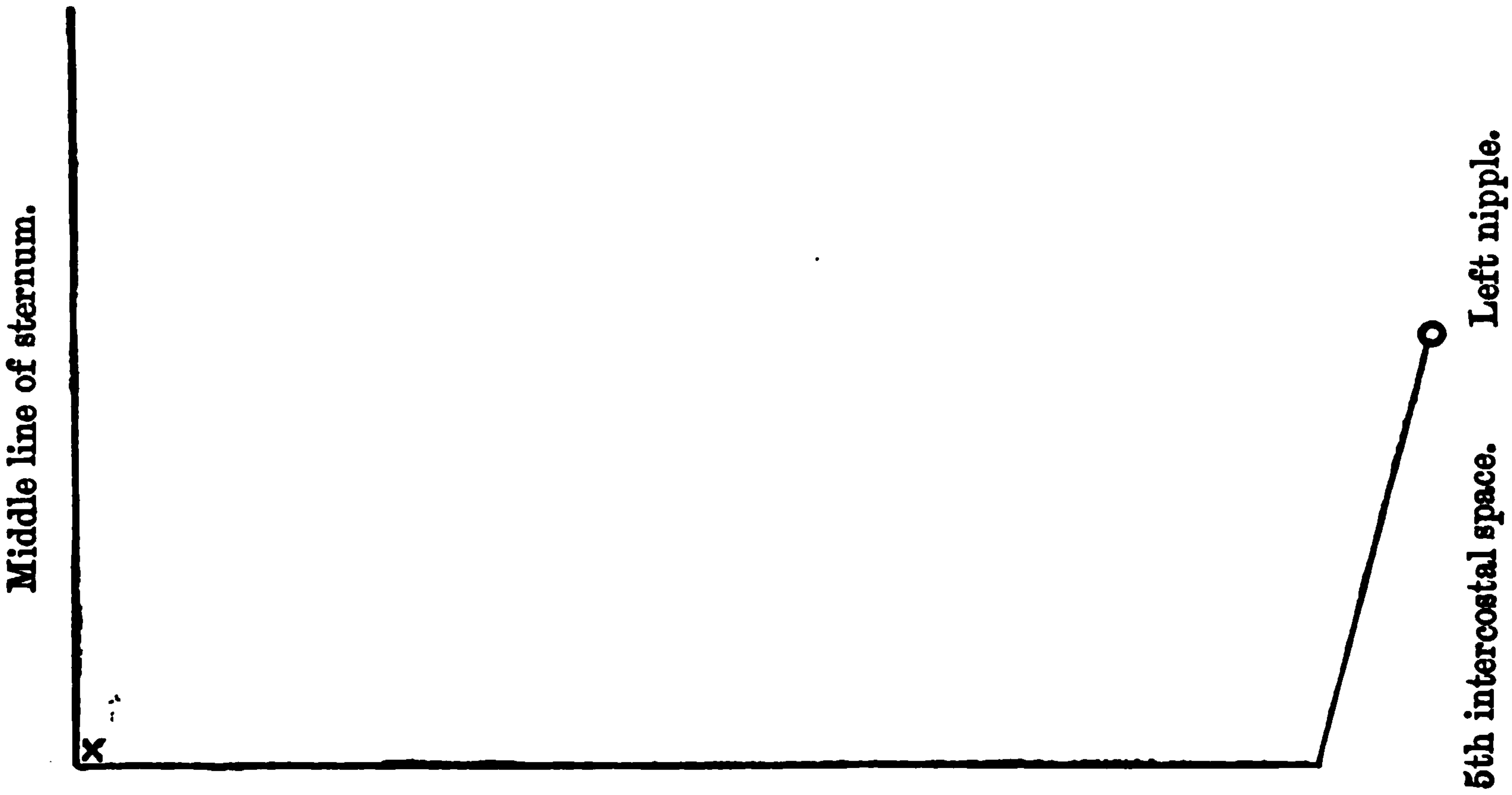
There was no mark of an entrance wound to be found on the chest, but my house surgeon, Mr. Brewer, drew my attention to a very slight fulness in the fifth intercostal space, about the existence, however, of which some who examined the chest were uncertain, so slight was it. On passing my finger very lightly over this spot, I thought that a slight elevation could be felt, and although great doubt was expressed on this point yet in consideration of the man's story, and knowing that the incision through the integument could do no harm, I decided to explore and so settle the question.

Having selected a spot at which it felt as though with each impulse of the heart something firmer than the surrounding tissue was pushed against my finger, moving with the impulse, and if a foreign body, fixed it seemed to me, in the heart, I found that the point thus chosen was one inch and two tenths below the left nipple, three tenths of an inch to the mesial side of a line perpendicular to the nipple, and three inches and five tenths from the middle line of the sternum (Fig. 1). It corresponded with the fifth intercostal space.

The heart's sounds were natural and the impulse was not increased.

Chloroform having been given to the patient, who had been moved into the operating theatre, the skin and subcutaneous structures were divided, the latter being deeply laden with fat, and then the end, the broken eye as it proved, of the needle, was distinctly felt. Clearing away the cellular tissue which lay immediately over the foreign body, its extremity, on a level with the surface of the intercostal muscle, was brought into view. It moved with each

FIG. 1.



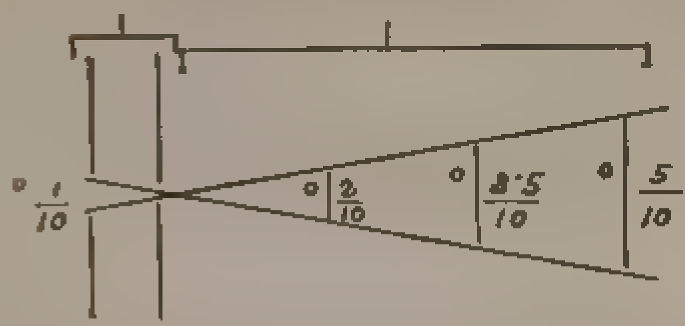
× One inch above the extremity of the ensiform cartilage.

impulse of the heart, describing a curve forwards, and at the same time a second curve to the right, the latter being evidently caused by the moving of the opposite end of the needle to the left with the action of the heart. The space traversed by the exposed end of the needle, vertically, horizontally and forwards, was in each direction by measurements (with callipers) just under one tenth of an inch.

After measuring the range of motion I held with ordinary forceps and drew out from the chest (in a vertical line as the patient lay on the operating table) a needle entire, with the exception of the extremity of the eye, and one inch and nine tenths in length. The heart's action was not affected during or by the withdrawal of the needle, but some blood welled up from the puncture, and continued to ooze until the wound was closed by sutures, and covered with a compress of lint soaked in carbolised oil.

The accompanying diagram shows the length of needle in the wall and also in the cavity of the chest. It gives the extent of movement at the eye and, allowing for the thickness of the intercostal wall and for the length of the foreign body, it indicates the range of movement within the chest (Fig. 2). The measurements figured in the diagram

FIG. 2.—Length of needle.



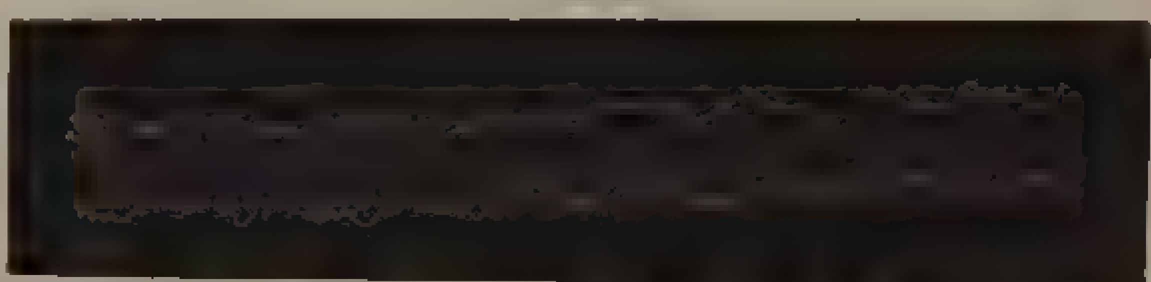
o Minimum range of vertical and horizontal displacements.

are based on the supposition that the needle worked on a centre corresponding with the inner surface of the wall of the thorax, and therefore give the minimum range of vertical and of horizontal displacements.

From the time of his recovering from the effects of chloroform the patient was entirely free from pain, and, with the exception of certain variations in the frequency of his pulse, convalesced without any marked symptoms. I considered it prudent to keep him in bed until time had been given for the repairs of the structures which had been punctured and he was not allowed to get up until four weeks had elapsed. On the fourth of December he was up for the first time but almost immediately experienced a severe pain which he described as being deeply seated under the left nipple and being alarmed he again lay down. After a few days he was able to be up and about, and on the 20th of December was discharged to the Convalescent Hospital.

The day after the operation Dr. Lauder Brunton had the kindness to take a sphygmographic tracing of the radial pulse, of which the accompanying is a copy (Fig. 3).

FIG 3



Tracing of radial pulse. Oct. 29th, 1872.

The notes of the patient's pulse, temperature, and respiration after the operation are given in the following table.

Day after operation.	Pulse.	Temp.	Resp.
Evening	72	98·2	18
1st day.—Morning	92	99·5	25
„ Evening	64	98	27
2nd day.—Morning..............	76	98·2	22
3rd day „	76	98·2	22
4th day „	76	98·2	22
5th day „	88	98·8	18
„ Evening..............	64	97·2	14
6th day.—Morning..............	64	97·2	16
7th day „	64	97·2	16
12th day „	70	98·5	16
13th day „	62	...	...
14th day „	70	...	...
15th day „	56	...	...
16th day „	68	...	...
17th day „	58	...	...
18th day „	68	...	..
21st day „	68	...	...

There are only a few words I would add to the history of this case.

(*a*) The pain of which complaint was made was due one must suppose to irritation of an intercostal nerve. It was referred so accurately to the course of the intercosto-humoral and to the lesser internal cutaneous, that no other explanation of it can be entertained.

(*b*). The two scars which are seen on the patient's chest are due to my first mark having been moved from its relation to the intercostal space by the raising of the patient's arm by an assistant just before the operation was commenced, a movement which escaped my notice at the moment, but which rendered it subsequently convenient to make the second and lower cut.

(*c*). The needle must have passed through the heart close to the apex. The movements communicated to it were too regular and agreed too well with those of the impulse to have been caused by anything but the action of the heart upon the needle which transfixed it.

(*d*). Two cases have been recorded in which it is said that the removal of a needle from the heart has been followed by recovery. The reports, of these cases are, however, unsatisfactory. Of the first (Case 10 in the appended table) it is said that the patient had all the symptoms of acute inflammation of the heart and of the pericardium, yet these symptoms disappeared the instant that the foreign body was removed. This is, at least, a very improbable story. All that is told us respecting the position of the needle is, that it was found "in a line with the external intercostal muscle." In the other case (No. 12 in the table), there is no evidence that the needle had wounded the cardiac walls or the pericardium. It had entered in the sixth intercostal space and was therefore, in all probability, below the situation of the heart.

(*e*). It is unfortunate that Dr. Fischer's table of cases of foreign bodies in the heart and Pericardium has been translated and published in the 'System of Surgery' without its having been verified. In taking from it the cases from which it may be inferred that certain patients have survived for some time the introduction of a needle, or some similar body, into the heart (the foreign body being extracted by a surgeon or remaining within the chest), I find, on looking to the original reports of the cases, so much that is doubtful and such inaccuracies in transferring them as alter very much the complexion of the table. I append a list of these cases, giving with it the numbers which refer to Dr. Fischer's table, and by comparison the important differences in the two will be manifest.

(*f*). The material point is this—that so far as these bodies, needles and the like, are concerned, there are only three cases on record (Nos. 1, 11, 13, in table), the original reports of which are the only ones I have been unable to refer to, in which it is possible that life has been preserved for more than a few weeks after the entrance of the foreign body into the textures or cavities of the heart. In all the remaining cases of supposed retention of life there is no evidence as to the time at which the heart was injured.

There is simply presumptive evidence that it was reached, if at all, through the œsophagus or stomach.

(*g*) These remarks apply only to cases in which needles or similar bodies have been found in the heart, to which class of cases alone my present observations refer.

Cases in which needles or similar foreign bodies have been removed from or found after death in the heart and pericardium, and from which it has been inferred that life may be retained for a long time after the occurrence of the injury. Evidence doubtful in most cases.

No.	No. in Fischer's table.	Foreign body.	External entrance.	Situation in heart or chest wall.	Duration of life.	Reference.	Remarks.
1	2	Needle	Sternum	Entirely in the wall of right ventricle	6 years	Gerard, Essai sur la Lethalité des Plaies Pénét. du Cœur. Thèse, Strasbourg, 1858, Obs. 7	Quoted by Fischer.
2	4	Needle	No evidence	Entirely in the wall of left ventricle	No evidence	Niel, New York Jour. of Medicine, July, 1849	Subject brought in for dissection. No opening in heart by which it could have entered at the time of injecting the subject for the rooms. Condition of adjacent parts not well made out. Subsequently ascertained that he had worked for twelve months since entering hospital in 1847. No evidence as to time of entrance of needle.
3	10	Needle	Œsophagus	In the wall and cavity of both ventricles	22 days after beginning of heart symptoms	Leaning, Philadelphia Medical Examiner; Lond. Med. Gazette, Jan., 1844	Dr. Leaning says that this needle in nine months traversed the pleura, lung, diaphragm, œsophagus, and finally entered the heart. It was originally introduced one inch below and on sternal side of left nipple.

4	12	Needle	Œsophagus ?	In the wall and cavity of right auricle near vena cava	No evidence	Sklersky, Ami de la Santé, 1841; Jamain, des Plaies du Cœur, p. 45; Legrand de Saille, Gazette des Hôpitaux, 1858	A woman, over 50, had suffered from pain in the chest for a long time. She died suddenly at the time at which an aneurism burst into the pericardium.
5	13	Pin	No evidence	In the wall and cavity of left ventricle	No evidence	Darian, Report by Legrand de Saille, Gazette des Hôpitaux, 1858	Patient died from erysipelas. No history or symptoms relating to the pin which was found at the post-mortem examination.
6	20	Thorn	Œsophagus	In the cavity of right ventricle	1½ year	Kussmaul, Würzburg Med. Zeitschr., 1864	Quoted by Fischer. No evidence as to time at which heart cavity was reached.
7	22	Needle	No evidence	In the cavity of left ventricle	No evidence	M. Pierre for Laugier, Bulletin de la Société Anatomique, p. 334, 1848-49	No history as to mode of entrance. No symptoms. Embolism of arteries and gangrene of leg.
—	23	—	—	—	—	—	Quoted by Fischer. The same case as No. 22.
8	29	Fish-bone	Stomach	Uncertain	No evidenee	Med. Times, vol. i, p. 467, 1860	Newspaper report of coroner's inquest. Mr. Andrews found a fish-bone protruding through stomach into heart.
9	30	Needle	No evidence	Uncertain	No evidence	Hennen's Military Surgery, Edin., 1818, p. 429	The patient suffered from symptoms of carditis for three months.

No.	No. in Fischer's table	Foreign body	External entrance.	Situation in heart or chest wall.	Duration of life.	Reference.	Remarks.
10	40	Needle	5th intercostal space	Uncertain	No evidence	Lynch O'Connor, Trinidad, Lond. Med. Gazette, Oct., 1835, p. 82	No evidence in report of wound of heart. Needle found "on a line with the external intercostal muscle." Symptoms: "Hurried respiration, and all the symptoms of acute inflammation of the heart or its membranes," which subsided the moment the needle was withdrawn. Patient recovered.
11	43	Iron pin	Thorax	In sternum, lesion of base of heart	12 years	Krügelstein, Henke's Zeitschr., 1843, 4, s. 295	Quoted by Fischer.
12	44	Needle	6th intercostal space	Uncertain	No evidence	Trelat, Bulletin de Therapeut., 1845, tom. xxix, p. 558; also Legrand de Saille, op. cit.	No symptoms. The patient, a lady, complaining that the surgeon had left a needle in her chest, induced him to try and remove it. No evidence that it had passed into heart or pericardium. Patient recovered.
13	—	Needle	No evidence	In left ventricle, point penetrating into left auricle through cusp of valve	No evidence	Biffi, Gaz. Med. Italiana Lombardia, Oct. 16, 1869; Med. Times, vol. ii, 1869, p. 532	Death from gangrene of tongue. The only evidence was a statement by the friends that the patient had told them he had thrust a needle into his chest 22 months previously. The patient was a lunatic.

A CASE

OF

EXCISION OF THE KNEE-JOINT FOR DISEASE

IN A WOMAN FIFTY-THREE YEARS OF AGE, WITH SUCCESSFUL RESULT.

BY

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Received February 4th—Read February 11th, 1873.

THE object of this paper is to lay before the Society the particulars of a case wherein excision of the knee-joint was performed for the result of a disease not hitherto but in rare instances submitted to the operation, namely, chronic rheumatic arthritis, and at the most advanced period of life that excision has hitherto, I believe, been practised, or at least recorded, the patient's age being fifty-three years. This case is, therefore, a notable exception to the typical conditions of disease appropriate for excision, which I brought forward in a previous communication, published in vol. liii of the Society's 'Transactions.'

Sarah A—, æt. 53, residing at Dunstable, was admitted into the Royal Free Hospital, July 25th, 1872. The history of her present state, as taken from the report of Dr. M'Kellar, Senior Resident Medical Officer, is briefly as follows:

Twenty-three years ago she had an attack of rheumatism, which, having affected various joints, finally settled in the left knee, giving rise to severe pain and considerable swelling,

which confined her to bed for ten weeks. Recurring arthritic disease lasted up to December, 1871, a protracted period of twenty years. Then she had a fall from some height downstairs, the leg on the affected side being twisted under her. This injury immediately superinduced a worse attack in the knee-joint, and she remained in bed again for nine weeks. Treatment was adopted without affording relief, and at length she came to be under my care at the hospital.

On *admission* the left knee-joint was much enlarged, measuring in circumference two inches more than its fellow; it had a globular form, but presenting some irregular nodosities, apparently of an osseous character around the articular ends of bone; the joint had undergone partial dislocation backwards, the head of the tibia projecting behind the condyles of the femur, and false or fibrous anchylosis had taken place, giving some degree of immobility to the articulation, although this depended partly on a generally thickened state of the ligamentous and aponeurotic structures, and on the ossific deposit around the joint. Thence the subluxation became more conspicuous when the patient, a very corpulent woman, attempted to stand on the limb, whereby the condyles of the femur abutted more prominently forward, showing that the anchylosis was loose and flexible. The patella was firmly anchylosed to the femur just above the outer condyle.

Considering this useless state of the limb, excision of the joint seemed justifiable, with the view of inducing osseous union, and restoring a useful member. But the continued recurrence of severe attacks of pain, affecting the patient's general health, and the otherwise incurable character of the disease, more immediately determined the propriety of operation; while the prolonged duration of the joint condition, the necessity for interference ere the patient's constitutional reserve-power might prove insufficient for recovery after operation, and the relatively advanced period of life, admitted of no further delay.

Accordingly I had recourse to excision, notwithstanding the patient's age.

The report continues. August 10th, Mr. Gant performed the operation by his usual method :—A semilunar incision being made from the outer condyle of the femur downwards to just above the tubercle of the tibia, and upwards to the same point on the inner femoral condyle the joint was readily laid open. The osseous anchylosis of the patella was sawn through, in order to raise that bone, and the femoral section made so as to remove about one inch and a quarter of the articular end of the bone; then the articular surface of the tibia was removed to the extent of about half an inch, this section corresponding to the margin of the incision. Lastly, the patella was dissected out. Hæmorrhage soon ceased, or was easily arrested as usual, by torsion of two or three articular branches, exposure and cold water. By straightening the limb the ends of bone were seen to be in easy, parallel apposition. The double splint Mr. Gant is accustomed to use was then applied. Any clots of blood were afterwards thoroughly sponged out of the wound, and finally the exact apposition of the bones having been again ascertained, the integument was laid down and the incision evenly closed by six or eight points of silk suture. Strips of lint, soaked in carbolic acid solution, were overlaid and to a sufficient thickness for a compress; this was encapsuled in oil-silk, and the whole retained by a small roller-bandage.

After the operation no unfavorable symptom occurred of consequence. Primary union went on, except, as usual, at the angles of the wound, which gave issue to a moderate amount of oozing serous discharge; and in three weeks the line of incision had become soundly united. In the course of a week or two the patient was allowed, by her own request, to sit up in bed, reclining on a back support. *Seven weeks* after operation the splints were removed; firm union of the bones was found to be established, with the limb, in a perfectly straight position. But the patient could not yet raise her leg from the bed. Apparatus reapplied.

October 4th.—The patient was carried from bed, and permitted to sit up for some hours, the limb in splints resting on two chairs. She was suddenly attacked with erysipelas,

which, commencing in the foot and leg, spread up the thigh, sparing the front of the knee. The whole limb became of a deep red or purple colour, and enormously swollen, tense and brawny, accompanied with great constitutional disturbance, so that for some weeks her life was in danger. The ordinary treatment was pursued, excepting not having recourse to incisions. Vesicles and bullæ formed and broke, and a persistent oozing discharge of serum gradually relieved the extensive infiltration, although some patchy sloughing of the skin ensued; and the erysipelatous inflammation extended further up above the buttock to the loins, and round to the lower part of the abdomen. Eventually the limb remained in a state of great œdema.

During the course of this attack, the splints having been necessarily removed, the limb gradually became slightly bowed outwards at the knee, yet the union remained firm, and the incision never reopened or gave issue to any discharge. Oxide of zinc was powdered over the whole surface, forming a kind of casement which absorbed the moisture, and seemed to stimulate its internal absorption. The œdema subsequently yielded to bandaging, aided by the horizontal position of the limb. At length, December 16th, a starch bandage was applied, and on the 21st, or little more than four months after operation, the patient went home to Dunstable, with instructions to return for examination in about a month.

State of the limb, January 24th, 1873, five and a half months after operation.—Union firm and immovable. The bowed appearance at the knee is corrected. Measurements of the limb are as follows:—*Length* 32½ inches, sound limb 35½ inches, or 3 inches shortening as the result of excision; *circumference* of knee 15½ inches, of sound knee 14½ inches, or 1 inch more after excision; the calf 12½ inches, and at the same level of the calf in sound limb 13 inches, or only ½ inch less after operation; the thigh 19½ inches, and at the same level of the thigh in sound limb, 21 inches, or 1½ inch less after operation. The functional use and power of the limb may be thus estimated at the

period referred to: the use of the limb for support in progression is not yet perfect; but the patient can bear her heavy weight upon it, and can walk with the aid of a stick; the power of the limb, according to its muscular development compared with that of the other limb, is indicated by the circumferential measurements of the calf and thigh. As the limb is brought into more active use there cannot be a doubt that it will become even more nearly equal in respect to its size and strength.

Proceeding to comment upon the particulars of this case, some of them seem to be more especially suggestive.

First, the kind of disease submitted to excision was, I believe, almost entirely exceptional to previous experience. In the histories of 100 cases collected by Mr. W. P. Swain, and which represent the majority of the cases operated on by Sir William Fergusson and some other surgeons of experience, no allusion whatever is made to rheumatic affections, except in two instances. Of one of these cases (No. 84) the briefest description is given—that it was "rheumatic disease of the knee-joint," of no specified deviation, that the result was recovery, but that of this case "no notes were taken." In the other case (No. 92) the patient had been attacked with "acute rheumatism" twenty years previously, but that "she used the limb until eighteen months ago, when she fell, sprained and struck the knee, and since that the limb has been useless." The result of operation was recovery with a useful limb. In a series of 39 cases of knee-joint excision by Professor Humphry, recorded in the 'Transactions' of this Society (1869), in no instance was the "nature of the disease," as described, in any way connected with rheumatism. A similar exclusion prevails in 12 cases by Mr. H. Lee, as recorded in the same volume of 'Transactions;' and also in the histories of 31 cases collected by Mr. Butcher, and in a second collection of 51 cases ('Operative and Conservative Surgery').

One case only of excision of the knee-joint has come to my knowledge, wherein the operation has been performed for chronic rheumatic arthritis; it was in the practice of

Mr. Curling at the London Hospital, and the result was successful ('Lancet,' 1869).

Secondly, the age of the patient in my case is, I believe, the most advanced period of life at which excision of the knee-joint has hitherto been performed. In Mr. Curling's case the patient's age was only 23 years. From the various series of cases already referred to I have gathered the most advanced ages at which the operation has been performed for other conditions of disease and the results.

It appears that excision of the knee-joint for disease, as performed in 13 cases between the ages of 32 and 47 years, has yielded 7 successful results; and that between the ages of 41 and 47 years there were 11 cases with 6 successful results. If then my own case, at the most advanced period of life, be superadded, we have a total of 12 cases, with 7 successful results, between the ages of 41 and 53 years. With regard to the question of mortality; in one of the unsuccessful cases of excision, amputation afterwards was followed by recovery, thus making a gross total of 8 cases out of 12, or an average of 2 recoveries in every 3 cases; and if the accidental deaths by embolism on the eightieth day, and phthisis, be omitted the recoveries rise to 8 in 10 cases, or 4 out of 5—a proportion fully equal to the successful results of amputation of the thigh for chronic disease of the knee-joint; with the advantage in favour of excision, that the limb is preserved.

Looking at all these cases of knee-joint excision for disease, two conclusions may be drawn; the one as to the extreme rarity of this operation for chronic rheumatic arthritis; the other that the operation may possibly be performed with safety and advantage at a much later period of life than it has hitherto been commonly practised. The result of the case I have brought forward would more especially suggest an occasional extension of the operation, both in respect to the kind of disease and the period of life which may be appropriate, thus embracing a larger class of cases beyond those hitherto recognised as suitable for excision.

SUCCESSFUL CASE

OF

GASTROTOMY IN EXTRA-UTERINE PREGNANCY.

BY

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Received December, 1872—Read February 11th, 1873.

Jane N—, æt. 29, applied at the hospital on September 23rd, 1872, to ask advice on the question of her being pregnant. She had been married two years and had menstruated regularly until about twelve months before her first visit, her periods lasting for about seven days, being rather profuse and perfectly painless. The subsequent cessation of the menses was quite sudden. From the middle of March, 1872, until about the 20th of July, she felt very distinct movements. At the latter date she felt "poorly" for a week, and there was a slight sanguinolent discharge. From that time no movements were felt. Nothing more definite than this can be obtained as to the occurrence of a "false labour," upon which so much stress has been laid by some writers for the diagnosis of extra-uterine gestation after the term.

On each side of the patient's chin, and in a line with the canine teeth, a well-pronounced tuft of hair existed, and she had a very distinct moustache.

The whole of the lower third of the abdomen was occupied by a tumour which was more prominent on the left than on the right side; at four spots (two on the left and two on the right side) irregular movable nodules were to be felt. The patient stated that two months ago, that is, in July and after the movements ceased, she was much larger than at the time of examination, the diminution having been gradual. The measurements were, on Oct. 4th—

	Inches.
Umbilical girth	36
Xiphoid cart. to umbilicus . . .	12½
Umbilicus to pubis	6
„ right anterior spine of ilium .	7½
„ left „ „ .	8

The uterus was rather larger than normal being three inches in internal measurement, soft, somewhat fixed, the cervix quite open, and the organ did not move with the tumour. Behind the uterus was a hard semilunar ridge, prominently felt in the rectum. Neither abdominal nor vaginal auscultation gave any sign.

The diagnosis of the case was easy and subsequently was found to be exact, to the effect that it was a case of retro-uterine gestation, and that the placenta was probably attached to the fundus uteri and left broad ligament.

I summoned a consultation of my colleagues on October 5th, and as they agreed to my proposal of removing the dead child by abdominal section, I performed the operation on November 2nd, the patient being under the influence of chloroform. I opened the peritoneal cavity as for ovariotomy, and found that the roof of the sac joined the parietal peritoneum about three inches above the pubis. I opened the sac, cautiously guarding against the flow of the contained fluid into the peritoneum by a sponge, and came upon an arm. Extending the incision upwards, I found the legs and brought them out, the trunk readily following. I had much difficulty in removing the head, for it was packed deeply into the pelvis, was very much distorted, and the skin and hair had become adherent to the floor of the cavity. Indeed,

a great deal of hair was left and had to be picked out subsequently during the gradual closing of the sac. The vaginal ridge I have spoken of was the edge of one of the parietal bones. I found that the placenta was attached to the posterior surfaces of the uterus and of the left broad ligament. I cut the cord, which was quite shrunk, about midway, and left the placental part hanging out of the wound. The placenta I left untouched. I sponged the cavity carefully out, and then by catgut ligature I stitched the cut edge of the sac all the way round to the peritoneal edge of the parietal wound, by a continuous tailor's stitch, in this way completely closing the peritoneal cavity. I then closed the upper half of the abdominal wound by deep silver sutures. The length of this wound was nearly seven inches, it having extended rather more than an inch above the umbilicus.

There was some sickness after the operation, which was checked by ice and champagne. I inserted a syphon deeply into the pelvic cavity, and by that means kept up for several days a discharge of offensively smelling fluid; subsequently I had the cavity syringed out every eight hours with a solution of sulphite of soda. About the 10th of November (the eighth day after the operation) healthy pus was discharged from the wound, and this continued with more or less admixture of placental *débris* during the slow contraction of the cavity. Occasional shreds of placenta appeared when the cavity was syringed out (night and morning), until November 29th, when I removed several large masses which were loosely attached to the left broad ligament, and also a quantity of hair. The pelvic cavity was completely closed early in December, and now (December 30th) the upper cavity is quite closed and nothing of the wound remains but a small sinus.

This is the second case in which I have operated, the first having been unsuccessful from the removal of the placenta. The success in my second case I attribute entirely to leaving the placenta, to closing the peritoneal cavity and leaving that of the abnormal sac open, having therein followed the successful practice of M. Koeberle of Strasbourg.

The struggle with hectic which the patient kept up will be

seen by the temperature chart (Plate IV); so that the operation is one after which the patient must be prepared to battle for life. On the whole, however, I certainly think it one which ought to be undertaken, before the child dies if possible, and always if the child be dead. The chances of the mother are quite as good as in ovariotomy, and, unlike those of the latter operation, can never be improved by delay. The removal of the fœtus before the absorption of the liquor amnii ought to be the rule; for there can be no doubt that the adhesions that may be contracted between the child and the cyst are formidable complications.

DESCRIPTION OF PLATE IV.

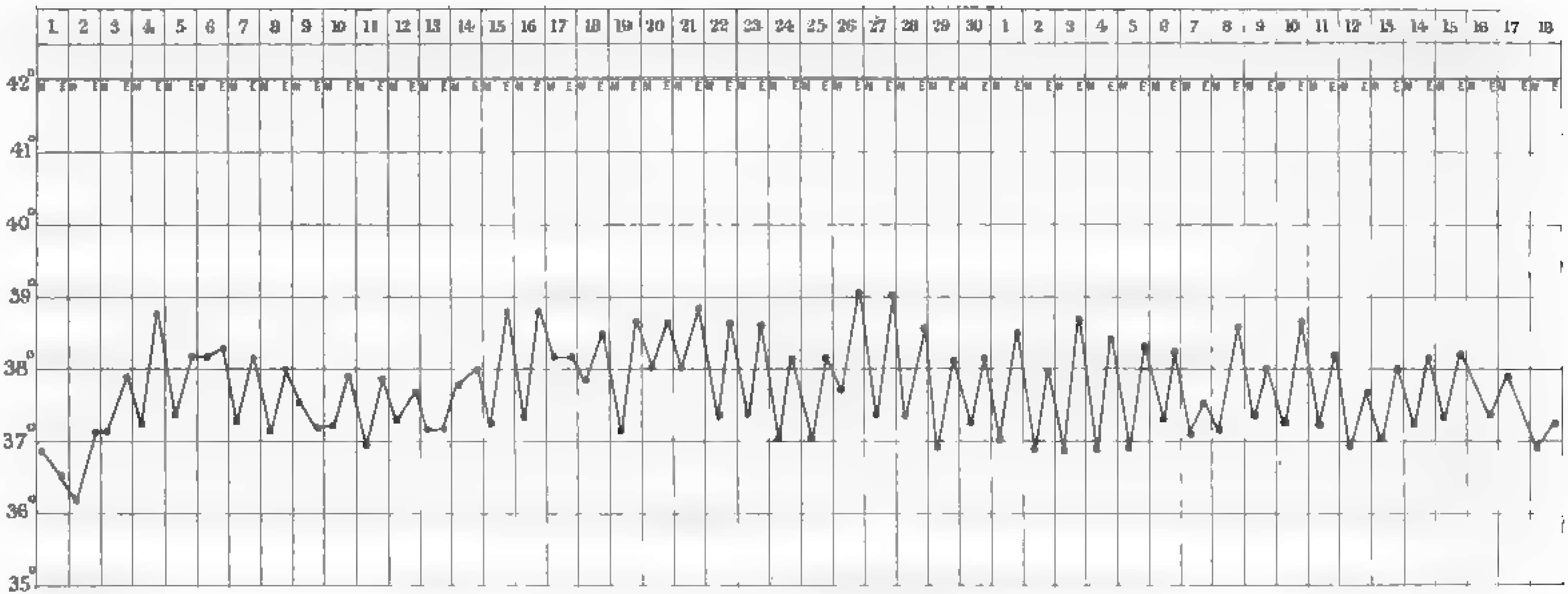
Diagram of temperature in a case of Gastrotomy performed by Mr. Lawson Tait in November, 1872.

TEMPERATURE CHART.

TEMPERATURE, CENTIGRADE SCALE.

Ranging from 95. to 107.6° Fahrenheit.

Gastrotomy performed Novr 2nd 1872. *Name Jane N.* *Age 24* *Result. Recovery*



G H Ford.

ON

DISSEMINATED SUPPURATION OF THE KIDNEY,

SECONDARY TO CERTAIN CONDITIONS OF URINARY DISTURBANCE.

BY

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Received January 28th—Read February 25th, 1873.

I PROPOSE to describe the peculiar suppurative condition which is sometimes spoken of as the "surgical kidney," and is produced, as I shall presently show, by the contact of unhealthy urine. Of all renal disorders, next to the varieties of albuminuria, it is the most destructive to life. It may almost be said to form the natural termination of stricture of the urethra, and is the especial danger which attends the use of the catheter and lithotrite. The attention which has been directed to this affection, however, is scarcely commensurate with its importance.

The post-mortem appearances, such of them at least as are evident to the naked eye, were well described and depicted by Rayer* under the term pyelo-nephritis. He associated the disease with inflammation of the pelvic mucous membrane, which as he thought produced a similar change in the kidney by extension along the excretory ducts. This view has practically held its ground to the present time. Sir Benjamin Brodie,† who seems to have regarded the disorder in a different

* 'Maladies des Reins,' 1841, vol. iii, p. 241.

† Brodie on 'Diseases of the Urinary Organs,' 1849, edit

light, has described the pathological appearances in these cases with all his wonted accuracy. He speaks of the vascularity of the kidney, of the deposits of yellow lymph and the small abscesses scattered through it, of the offensive mixture of pus and urine in the renal outlets with phosphatic precipitations on the pelvic membrane, and refers to the constant association of these conditions with cystitis; but strangely for so careful an observer he appears to have inverted the morbid relation which we must believe to hold between the kidneys and the urinary receptacles, and regarded the suppurating state of the kidney as the cause instead of the consequence of the vesical disturbance. This view appears to have met with little acceptance. The condition has since been occasionally referred to by practical surgeons, among whom I may mention Mr. Charles Hawkins,* who connects it with the presence in the bladder of decomposed and ammoniacal urine. The process has been generally regarded with Rayer as the spreading of inflammation from the bladder to the kidney by continuity of structure. Dr. Wilks† alone, who has been more precise than other writers, describes this variety of renal suppuration as connected not only with inflammation of the bladder, but with purulent absorption. From the bladder, as he says, "the inflammation extends up the ureter to the pelvis of the kidney; thus an absorption of the purulent elements takes place into the tissue (a more probable process than an extension of the inflammation up the straight tubules), and a suppuration occurs in the cortical part of the organ."

My inquiries, which were made some time ago, though hitherto unpublished, are pathological rather than surgical in their design. Where I trench upon the outskirts of surgery and tread upon ground foreign to my habitual experience I must ask for the indulgence of the surgical Fellows of the Society.

The change usually affects both kidneys, though in rare cases it is limited to one. The pelves are generally more or

* 'Holmes's System of Surgery,' article on "Lithotrity."

† 'Lectures on Pathological Anatomy,' p. 358.

less dilated, and give evidence of inflammatory action in the state of their mucous membrane. This is commonly injected, often thickened, creamy, and suppurating, frequently encrusted with phosphatic salts, and not seldom partially or superficially sloughing. The kidney, if the primary obstruction be, as it generally is, of considerable standing, shows the results of wasting pressure and chronic vascular disturbance. The cortex is thinned, the capsule is thickened, and holds with unnatural tenacity both to the glandular surface and more closely still to the surrounding fat, which itself is often markedly increased, probably in consequence of undue vascular injection around the kidneys. Disseminate renal suppuration may, however, take place without any preceding chronic change, though inflammation or congestion of some part of the urinary mucous membrane, possibly of recent date, appears to be an invariable antecedent.

The glandular condition nearly resembles to the naked eye, and also, as will presently appear in more minute respects, the renal manifestation of general pyæmia. The kidney becomes swollen and full of blood, much of which remains fluid after death, though some vessels contain clot evidently of ante-mortem origin. The tissue is variegated with blotches and streaks of intense injection; it is soft, friable, discoloured, and prone to decomposition.

The cones usually display to the scrutinising eye sharply defined white lines, which start from the tips of the mammillary processes in the pelvic cavity and pass into or through the cones in the direction of their striation. These streaks look like, what indeed they are, distended tubes, and are important witnesses of perverted action. Close to them swollen blood-vessels are often conspicuous.

Next, or possibly without such evidences of chronic change as the swollen tubes give, appear small, softly defined, fawn-coloured patches which streak the cortex from cones to capsule, or take the shape of wedges with the base against the capsule, the point entering the medullary tissue. These are at first scarcely less hard though more friable than the natural substance of the organ. They are surrounded

intermingled with vascularity. As the disease progresses they become large, irregular, and confluent, soften in their centres into ordinary liquid pus, and finally take the shape of scattered abscesses, varying from mere points up to the size of peas or even larger. These are often thickly sprinkled through the gland, occupying the cones, where they keep more or less of a linear arrangement, and are less regularly but more numerously distributed through the cortex. They appear on the surface sometimes as purple blotches in which suppurative centres can be seen, or as discrete or confluent pustules, often nearly resembling a cutaneous pustular eruption.

With this condition the microscope shows more or less dilatation of the straight tubes, distension or morbid occupation of the associated blood-vessels, and disseminated intertubular suppuration, the distribution of which is regulated by the course of the veins.

Passing to detail, and taking the straight tubes first as the parts of the organ first affected, these as they converge upon their outlets are often strikingly dilated apparently from the backward pressure of the retained urine. The dilatation is irregular, widening the cylindrical shape of the tubes or converting them into ovoid, globular or shapeless cavities. They are variously occupied by saline matter, purulent secretion, fibrin, or epithelial growth (see Plate V, figs. 1 and 2). The straight tubes, thus stretched and filled, form the white lines which are evident to the naked eye. The change does not extend to the convoluted tubes, which remain for the most part natural.

The veins, which appear to be next involved in the disease, are generally distended with blood. The straight veins of the cones often display in section a partial distension which is probably the result of coagulation which has occurred during life (see Plate V, fig. 3, and Plate VI, fig 1).

The larger veins of the cortex are often similarly filled, and it sometimes happens that the arteries which pass in companionship with them are likewise permanently occupied (see Plate VI, fig. 2). As a general rule, however, the arteries are natural, as also are the Malpighian vessels.

The venous position of the clot in the condition under consideration differs from the similar result of ordinary pyæmia, in that the obstruction there is essentially arterial.

The third stage of the disease is the scattered suppuration which is the most obvious result of the complicated process. The disseminated abscesses, or regions of cellular infiltration antecedent to abscesses, are intertubular, and have relation to the course of the veins. Cells gather at isolated spots, sometimes obviously accumulated around a minute distended vessel. Occasionally the new formation overspreads considerable districts, insinuating itself more or less evenly between the tubes, its vascular origin being chiefly evinced by its obvious intertubular position (see Plate VI, figs. 3 and 4). The Malpighian bodies remain unaffected by the disease, though the adventitious corpuscular formation often collects abundantly outside them. The convoluted tubes are generally clear, though where they cross the districts of infiltration their epithelium is sometimes superabundant, and sometimes they appear to be encroached upon, or confused by, the cellular formation around them. They are sometimes displaced or compressed.

Taking the structural changes in their mutual relation, the dilatation of the tubular exits, the morbid occupation of the veins, and the general absence of signs of tubal inflammation, the nature of the process is clear.

This disorder has its origin in the regurgitation of urine charged with morbific products. This, forced backwards by the retention general in these cases, distends or occupies the straight ducts. Thence by transudation, or similarly, it enters the neighbouring blood-vessels, and charges them with an infection resembling in its results that of pyæmia. This is distributed by the veins to the rest of the gland, sowing abscesses in their course, and ultimately causing constitutional symptoms analogous to those of pyæmia otherwise derived.

The condition of the kidney may be described as one of pyæmia arising within itself. It has a close general resemblance to that caused by a distant infection, differing from

it in the usual dilatation of the urinary outlets, and in the fact that, while with pyæmia from a remote source the *materies morbi* is necessarily distributed by the arteries, in the condition under discussion it is scattered by the veins into which it was first received.

The disorder in its frequency and fatality has great practical importance. Inflammation of the bladder, or of the pelvis of the kidney, either as antecedent to the change, or associated with it, is so invariably present as to give a seeming warrant to the old view which regarded the disease as a mere extension by contiguity of inflammation beginning in the urinary cavities. The nature of the organic change, however, plainly declares its origin, not in the mere creeping of inflammation from membrane to gland, but in the absorption of morbific matter. Of this the urine is obviously either the source or the vehicle. It remains to inquire whence and in what circumstances the poison is engendered.

To help in answering these questions I have collected the particulars of 69 cases of the disease from the post-mortem books of St. George's Hospital. The following table shows the urinary disease upon which the suppurative condition of kidney followed.

Disease antecedent to disseminated suppuration of kidney in 69 cases.

Obstacle to escape of urine.	Stricture of urethra	19
	Disease of prostate, enlargement, tumour, or abscess	12
Loss of expulsive power.	Paralysis of bladder from fracture of spine ...	5
	„ „ disease „ ...	3
	„ „ „ of cord ...	4
	„ „ „ of brain ...	3
	„ „ consequent upon exhaustion from disease or accident ...	2
Vesical calculus.	Stone in bladder, no operation	6
	„ „ lithotrity	6
	„ „ lithotomy	3
Cystitis from other causes.	Cystitis from vesical growths, &c. ...	3
	„ unexplained	1
	„ from discharge of lumbar abscess into bladder	1
Complicated.—Stone in kidney, with enlarged prostate ...		1
		69

I have next classified the descriptions of the urine in each case. The state of this secretion was noted in 47 of the number, in the following terms:

State of the urine in 47 of the cases previously referred to.

Reaction stated.	Ammoniacal or fœtid, and mixed with various products of vesical inflammation ...	21
	Ammoniacal	1
	Alkaline and turbid, or containing mucus ...	3
	Alkaline, bloody, and purulent	1
	Alkaline or "phosphatic"	3
Reaction not stated.	Ropy, containing mucus, pus, and blood ...	1
	Containing mucus and pus	1
	Purulent and bloody	4
	Purulent	6
	Bloody	4
	Albuminous and turbid, or purulent ...	2
		47

Looking first at the urine as directly connected with the origin of the disease it appears that three conditions of this secretion usually concur—retention, ammoniacal decomposition, and admixture with the products of mucous inflammation. Of these an essential circumstance appears to be ammoniacal decomposition, which retention may induce and cystitis either precede or follow. The urine was generally fœtid and more or less mixed with vesical products, pus, mucus, and blood. There is reason to believe that it was invariably ammoniacal. Wherever the reaction was stated it was persistently alkaline except in one instance. In this exceptional case the secretion was alkaline when the inception of the disease was declared by rigors, then for a short time acid, and alkaline again before death.

In the cases, comparatively few, in which the reaction was not stated, the condition of retention or the state of the urinary mucous membrane was generally such as to point unmistakably to ammoniacal change. The simple presence of pus or mucus in the urine, though lasting for years, does not appear to set up the renal disorder so long as the urine retains its acidity and resists putrefaction.

I have myself known no instance in which the mischief has arisen except in connection with ammoniacal urine.

On this point I appeal to the more ample experience of the surgical fellows of the society.

The ammoniacal change, however, though it may arise independently of mucous inflammation, produces it so constantly that the origin of the disease is always thus complicated. Sometimes, as with stone, the inflammation of the bladder has led to the change in the urine; sometimes, as in cases of paralysis, the change in the urine has caused the inflammation of the bladder. Whichever comes first, so long as the necessary foulness of the urine is attained, a condition which is promoted by the admixture of diseased vesical secretion, the renal suppuration may follow. Whether primary or secondary the inflammation of the urinary mucous membrane is invariable. This is usually of the bladder, though the renal change has been known to follow inflammation and retention confined to the pelvis caused by a stone situate in that cavity.

Passing from the state of the urine to its clinical antecedents they may generally be stated to be of three kinds: obstacles to the escape of the urine, loss of expulsive power, and vesical or very rarely pelvic irritation.

Stricture of the urethra is of all causes the most common, giving rise as it does to the needful urinary putrefaction, and in its chronic form ensuring the dilatation of the glandular exits which makes them ready recipients of the poison.

Enlargement of the prostate, scarcely less common as a cause of the disease, acts in the same way.

Next in order of frequency to such impediments come the diseases and injuries of the nervous system by which the expulsive power of the bladder is destroyed. These, giving rise to retention and decomposition of urine, and its contamination by the products of vesical inflammation, cause changes in the same sequence as those which arise from stricture, but more rapid in progress. The [illegible] innervation in these cases hastens the [illegible] mucous membrane, which gives the [illegible] urine observed in such [illegible] severe renal mischi[illegible]

cerebral disease, and occasionally from the general prostration which follows from disease or accident not directly connected with the nervous system.

Lastly, severe vesical irritation, though unconnected with retention, may cause the same results. The disorder was traced to stone in the bladder in 15 of the 69 cases previously referred to. Though differently begun a similarly putrescent state of urine to that of retention is here in the end produced, contamination by mucous discharges which promote decomposition being apparently the incipient evil. Putrescence appears to be always present. In connection with the frequent origin of the renal suppuration from vesical stone its extreme rarity as a consequence of stone in the kidney is worth remarking. The difference probably lies in the less putrefactive tendency of the discharges from the pelvic membrane or of the urine in this cavity.

As giving a deadly finish to the several causes to which the disorder has been ascribed, it is a matter of common experience that it has often been apparently lighted up in cases otherwise so tending, by catheterisation, lithotrity, and other mechanical procedures. The fact that the disorder is stigmatised as the "surgical kidney" bears witness to this liability. But, although the discredit to surgery involved in the term is not entirely without foundation, yet it would seem that this form of renal suppuration would be better distinguished by the term *uriseptic*, which would declare its general clinical relations more comprehensively.

Linking the secondary renal mischief with ammoniacal or putrid urine, and having regard to recent researches which have connected the lower kinds of organic life with pyæmia,* it is worth noting that the condition of urine which causes the disease now in question is one in which vibriones and bacteria abound; but considering the different circumstances in which such organisms appear it would be unsafe to draw any inference as to the nature of the virus beyond the broad fact that it is associated with, and apparently dependent [illegible]ition of urine.

* [illegible]ical Transactions,' vol. xxiii, p. 303.

The frequency of the disease after the introduction of instruments may lead to a surmise—but in the present state of our knowledge to nothing more than a surmise—that the essential virus is capable of being conveyed into the bladder by their means.

In connection with the pathology of the disease I may briefly sketch the symptoms, drawing chiefly from the cases to which I have already alluded. The disorder, particularly when the abscesses, as is too often the case, are widely disseminated, runs a rapid and fatal course. The patient generally dies within three weeks of the first symptom, sometimes within a few days. In 14 cases in which the dates and symptoms were carefully recorded in the hospital books the duration of the complaint varied from two to eighteen days. As an example of its rapid course I may refer to an old woman who was brought into the hospital with a simple fracture of the thigh. Two days after the accident she became unable to pass water. A catheter was used, and the urine found to be natural. It then quickly became offensive and loaded with mucus, and death occurred within a week of the accident, three days after the urine changed its character. Small purulent deposits were scattered through both kidneys.

The course of the disease resembles that of pyæmia, differing from it in the usual exemption of other organs from the suppurative process. The symptoms are general rather than local; they point to contaminated blood rather than to disturbance of glandular function. Pain in the loins, however, sometimes occurs; and often the urine is much diminished, or even for a time nearly suppressed. Shivering happens early and is apt to be repeated. Febrile symptoms rapidly follow with typhoid prostration. The pulse becomes rapid and feeble, the tongue dry and brown, the appetite absent. Vomiting is a frequent and often an urgent symptom. Not seldom hiccough occurs, and sometimes diarrhœa or profuse sweating. The countenance becomes anxious and haggard, the complexion cadaverous or yellow, and possibly with low delirium, the patient sinks into utter prostration, unconsciousness, and death.

Erysipelas is an occasional complication, as also is a condition of pulmonary congestion or œdema short of that which results in pyæmic deposits.

Dropsy is uniformly absent.

The disease is not always fatal. In examining the bodies of persons who have long suffered from disease of the urinary organs it occasionally happens that there are found upon the kidneys obvious scars, often much pigmented, in place and dimension such that they may fairly be attributed to ancient abscesses. Dr. Wilks tells me that he has made the same observation, and a case of the sort was related by Dr. Moxon in the last volume of the 'Pathological Transactions.'

As complicating the less rapid forms of the disorder must be mentioned perinephritic abscess. This results from perforation of the capsule of the kidney by a superficial pustule and its discharge into the areolar tissue.

In a body recently examined at St. George's Hospital a pint of pus lay outside the suppurating kidney; and preparations in the museum of St. Mary's Hospital illustrate a case in which a vast collection of pus reaching from the diaphragm to the groin, pushing forward the bowels and infiltrating the lumbar muscles, had the same origin.

In the treatment of the disease it is sufficiently clear that our efforts must be directed rather to prevention than cure, and to this end, beyond cautious surgery, measures of two kinds suggest themselves. The first is the preservation or restoration of the acid reaction and consequent chemical stability of the urine; the next is the use of antiseptics. When the urine has become ammoniacal it is difficult to restore its acidity by medicine. I have been more successful in doing so with mineral acids than with vegetable; and with sulphuric acid more than with nitric or hydrochloric. I have had no experience of the introduction of carbolic acid or other antiseptics by injection into the bladder, but the pathology of the disease is suggestive of such experiments.

To sum up: the form of renal suppuration, which may be termed uriseptic, has its origin in ammoniacal and putrid urine, poison from which is conveyed by the veins into the

substance of the kidney, and thence infects the system; causing symptoms which are characteristic of blood disorder rather than of change limited to the kidney, and resemble those of pyæmia excepting in the respect that organs other than the kidney seldom share in the suppurative process. As this diseased condition, though not necessarily fatal, is, when developed, little obedient to medicine, our efforts must be directed towards the correction of the state of urine from which it springs.

DESCRIPTION OF PLATES V & VI.

Plate V.

Fig. 1.—Enormous dilatation of straight tubes, some of which contain fibrinous plugs. Longitudinal sections. The lower outline (fig. 1 *a*) gives the size of the tubes in health. Abscesses after lithotrity.

Fig. 2.—Enormous dilatation of straight tubes as seen in transverse sections. The sketch (fig. 2 *a*) represents a similar section (identically prepared and magnified) from a healthy kidney. Abscesses after lithotrity.

Fig. 3.—Obstructed veins among straight tubes, from a case of scattered renal abscesses after lithotrity.

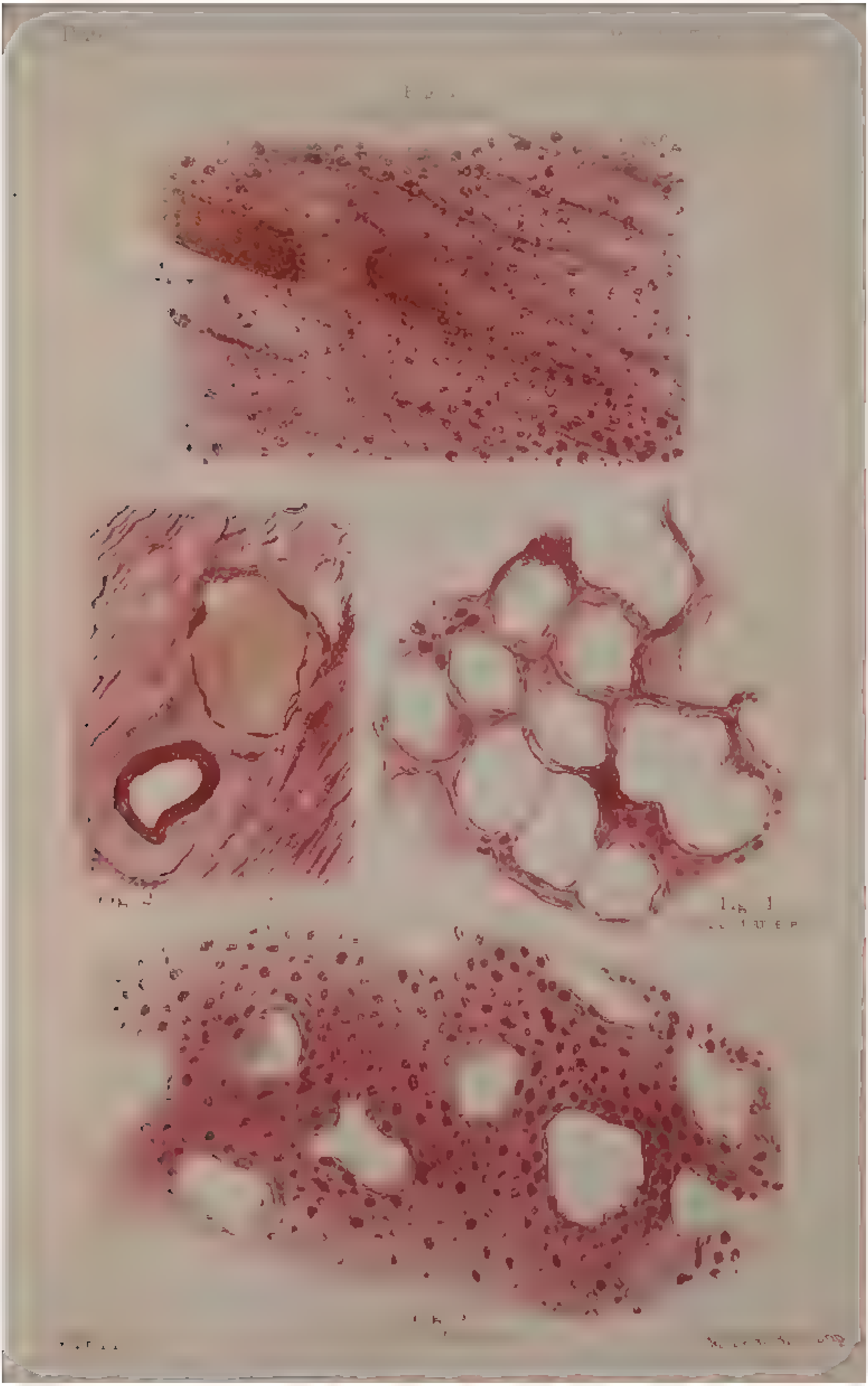
Plate VI.

Fig. 1.—Accumulation of corpuscles, preparatory to the formation of an abscess around an obstructed vein in cone.

Fig. 2.—Coagulum in a vein and not in its companion artery. From a case of scattered renal abscesses after lithotrity.

Fig. 3.—Matrix of kidney swollen by enormous production of corpuscles antecedent to abscess. The condition was circumscribed.

Fig. 4.—Ditto. The same tissue from a part of the organ which remained unaffected.



ON

THREE PECULIAR CASES

OF

MOLLUSCUM FIBROSUM IN CHILDREN

IN WHICH

One or more of the following Conditions were observed:

HYPERTROPHY OF THE GUMS; ENLARGEMENT OF THE ENDS OF THE FINGERS AND TOES; NUMEROUS CONNECTIVE-TISSUE TUMOURS ON THE SCALP AND OTHER PARTS OF THE SURFACE OF THE BODY, WITH VARIOUS SUPERFICIAL AFFECTIONS OF THE SKIN.

BY

JOHN MURRAY, M.D.,

ASSISTANT PHYSICIAN AND JOINT LECTURER ON PATHOLOGY AT MIDDLESEX HOSPITAL AND ASSISTANT PHYSICIAN TO THE HOSPITAL FOR SICK CHILDREN.

COMMUNICATED BY

THOMAS SMITH, F.R.C.S.

Received January 14th—Read March 11th, 1873.

I VENTURE to bring the subjects of the following remarks before the notice of the Society, because I believe they present a group of remarkable pathological phenomena not hitherto described in medical literature. It will not, I think, be necessary to relate the progress of these cases in any detail, since they have been under medical observation. Indeed the clinical history, although most important, may be told very shortly. It will be well, however, to describe at some length the local and general symptoms as they now exist, and as the report of the present condition of the eldest child will include a description of almost all the phenomena observable in the two younger patients, I shall,

to save repetition, confine myself in particular to a detailed account of her condition and point out in what respect the symptoms of the others agree or differ.

It is important in order that some clue may be afforded regarding the etiology of the affection that I should enter fully into the family history, and also refer to the sanitary conditions under which the patients have been placed.

Family history.—The patient's father and mother are cousins by their father's side. They keep a grocer's shop near Liverpool Road.

The father is illegitimate; he is a healthy-looking man, æt. 42. He is very temperate. A searching inquiry into his previous history elicits the information that he had an attack of what he called "inflammation of the chest" when 9 years of age; pains in the knee at 16; and that six years ago he was subject to a slight cough with loss of voice for six weeks. There is no evidence whatever of syphilis in his history or in his present condition. His father was he believes in every respect a healthy man, but he says that as he went to Australia shortly after his birth he cannot of course give much reliable information regarding him. His mother was afterwards married, but not to his father, and had eleven children, most of whom have died of consumption; her husband, however, and his family were consumptive. His mother is now sixty-eight years of age and remains healthy. His grandparents by his mother's side were all long-lived. There is no appearance whatever on his body of any skin or other affection. The mother, æt. 40, looks a healthy woman, and is now pregnant for the sixth time. She states that she has always been very nervous, and that immediately before and after marriage she was subject to great depression of spirits on religious matters, but this left after the birth of her first child. She has been subject to habitual constipation of the bowels. The catamenia have always been normal. There is no trace on her person of any similar affection to that of the children. There is no suspicion of her having been the subject of syphilis, and she presents no evidence of that disease. Her

father is 73 years of age and still earns his livelihood; her mother died, æt. 33, of consumption.

The only fact of importance, I think, which we have elicited in the family history is the near blood relationship which subsists between the parents. With the exception of the three patients and the grandmother, who is stated to have died of consumption, the family history is remarkably good. Both parents are and have been in the enjoyment certainly of average good health, and they afford no evidence whatever of a similar affection to that which afflicts their three children.

I shall have now to explain the circumstances under which the children were born, as it may prove to bear in an important manner on the question of the etiology of the affection.

The mother has had four children, also a miscarriage, occurring between the birth of the third and fourth children, The children were all born at or nearly at full time in the same house in John Street, Liverpool Road. The mother nursed them all. The eldest, a fine healthy boy, ten years old, was born and lived for eighteen months in the comparatively healthy *first* floor; the three others were born on the *ground* or basement floor. The mother was three months gone in pregnancy with the eldest affected child when the family moved to the damp residence. There was no kitchen or cellar beneath, and the sleeping apartment was, according to the positive statements of the parents, excessively and most sensibly damp, so much so that the mattress on which they slept became mouldy. They moved to another and better house, nearly two years ago, or six months after the birth of the last child.

The disease in one form or another was pronounced in all the three affected children when they left, and in a degree of increasing intensity corresponding with the age, markedly so in the eldest, and to a very slight extent only in the youngest. Since that time the affection has made more or less progress in all. I shall, however, recur more fully to this.

I shall now attempt to describe as faithfully as I can the history and present condition of the eldest affected child, in whom the disease is most marked. In her the skin and subcutaneous connective tissue, the periosteum, the gums, the ends of the fingers and toes, are the seats of disease. She is also deaf.

Ellen S— is seven years of age. So far as the mother was concerned the labour was apparently natural, but the medical man was obliged to have recourse to artificial respiration and other means to save the child's life at birth.

History of present illness.—The disease was first observed in the gums when she began to cut her teeth; they were, the parents thought, enlarged. The affections of the fingers, the neck, and nose were not noticed until the patient was about two years old, and the growth on the scalp not for a year afterwards. Her father says that she seemed to be deaf "at times," this symptom increasing with the disease. He adds that she has always been very shy in her manner; when two years and eight months old, the hypertrophied gum was partially removed by Mr. Erichsen at University College Hospital. The disease has continued to increase since the operation. When four or five years of age a tumour was removed by Mr. Wood from the scalp at King's College Hospital.

Present condition.—The child has a shy but not unintelligent appearance. She is a fairly nourished girl for her age and her complexion is pretty clear.

The face.—On her forehead, and encroaching in two cases on the scalp, are four tumours covered by skin and varying in size from a small pea to a date. They are smooth on the surface, firm, elastic, and pretty freely movable with the skin. The skin over the two largest is of a bluish tint, as if from venous congestion, that over the others natural. The growths are not painful, neither do they appear to be at all tender to pretty firm pressure (see Plate VII, fig. 1). The periosteum beneath one or two of these growths feels slightly thickened, but I am unable to satisfy myself thoroughly on this point. Both

on the lower aspect of the chin and the left cheek, near the border of the lower jaw, a similar tumour exists—that on the chin is about the size of a hazel-nut, and that on the cheek not larger than a pea.

The nose.—At the junction of the right ala of the nose with the cheek is a small and slightly raised, wart-like body, a *verruca plana*. In the same situation on the left side there is a commencing growth of a similar character. These growths are neither painful nor markedly tender.

The oral cavity.—The appearance of the gums is very remarkable. They are everywhere greatly hypertrophied, and they almost completely bury the teeth (see Plate VII, fig. 1). They form, in parts, numerous papillomatous or polypoid-looking growths, and in other situations present a peculiar fungating appearance; indeed, this latter characteristic of the growth is at once observed. Although, as I have said, the teeth are almost buried by the hypertrophied gum, their extremities are still in every case visible and are, in some measure, serviceable for the purposes of mastication.

The enlargement of the gums is most marked at their upper and free surface, where they are mostly flattened out, and in parts hardened, by the pressure of the opposing gum. They present the natural colour and, although they are in parts somewhat soft, vascular, and spongy looking, they mostly feel firm and fibrous to the touch. It is alleged by the parents that they have a tendency to bleed, but there is no evidence of this in her present condition. The mucous membrane in the oral cavity is elsewhere in every respect normal, the disease being distinctly limited to the gums. There is slight enlargement of the tonsils.

The ears.—Both ears present a curious appearance from the existence of prominent growths, chiefly on the anterior aspect of the *helix*. They number altogether about a dozen, and vary in size from a millet-seed to a bean. They are painless, and mostly hard, tough, and fibrous to the touch, and they are covered with pale, rough, and in parts glistening skin. Some of those on the posterior aspect of the ear are, however, softer, more elastic, of a bluish colour,

and apparently more vascular. The intervening skin is, here and there, the seat of small, variously shaped, pale and glistening elevations, not unlike vesicles at a distance, but painless, hard, and fibrous to the touch (see Plate VII, fig. 1).

The scalp.—On the scalp are four cicatrices, two above the right ear and one on either side of the occiput, the seats of previous tumours, similar to those on the forehead. In the neighbourhood of the cicatrix, on the left side of the occiput, there is some new growth. To the inner side of this is a small tumour the size of a hazel-nut. The growth in both these instances is apparently similar to that on the forehead.

The neck.—Extending rather symmetrically over the skin on the back and both sides of the neck, and spreading on to the back as far as the second dorsal vertebra, are about forty white and glistening flat elevations, like smooth warts, varying in size from a pin's head to a shirt-button; they are sometimes isolated and distinctly circumscribed, but others are found blended together in groups of about a dozen. The largest ones, at a distance, present exactly the appearance of vesicles, but they are hard and contain no fluid. The intervening skin is healthy. They are not tender or painful. There is one small wart on the skin in the right infra-clavicular region. Over the prominence of the seventh cervical vertebra, and extending above and below it, there is a tumour the size of a small chesnut apparently similar in character to those on the forehead. The growth is, however, softer and the skin is discoloured by ecchymoses.

The trunk.—The skin over both scapulæ, and extending over either axilla, is slightly ecchymosed, and on these regions are observed numerous and rather diffused growths beneath the skin, apparently of a similar kind to those of the scalp and forehead. On passing the hand over these affected regions the skin is felt to be indurated, raised, and nodular, or undulating, from the presence of somewhat hard growth, apparently neither painful nor

tender to moderate pressure. There is no affection of the surface of the trunk elsewhere.

The upper extremities.—The posterior aspect of the upper part of both arms presents a similar condition to that observed in the scapular regions. A small, hard, and symmetrical tumour, the size of a hazel-nut, exists on the outer side of either forearm.

The hands present one of the most peculiar features of the case. With the exception of the forefinger of the left hand, the superficial soft structures, at least of the last phalanx of the fingers and the nails, are greatly and curiously hypertrophied. This feature of the disease is entirely limited to the last phalanx. The enlargement, density, and appearance of the affected fingers are not at all uniform. The extremity of the finger is much more increased in size at one part than another; the surface is smooth in parts, elsewhere rough and nodular (see Plate VII, fig. 2). The smooth portions present here and there a cystic appearance, as if from dilated ducts filled with clear fluid, but on pricking these blood alone presenting no abnormal microscopical appearance escapes, and is not followed by local collapse. Other portions are transparent, of a bluish-red tint and vascular looking, with dilated vessels on the surface not unlike nævus at first sight, but the resemblance fades on closer inspection. The hard and irregular portions appear to be composed to a large extent of hypertrophied epidermic structures, which here and there may be peeled off. Firm pressure on the enlarged finger-ends causes the child to wince, but she does not generally appear to suffer pain from the affection. The nails are enlarged to a corresponding extent with the soft structures and are marked by numerous transverse furrows. The nail of the forefinger of the left hand, however, which is not clubbed as the others, is also healthy and free from furrows. It is interesting to observe that the corresponding finger of the right hand, although it has not escaped the enlargement, is much less affected in this way than the others. Indeed, a symmetrical character is observable in the affection of the hand

indeed, of the whole disease in the body. The unaffected forefinger, free as it is from the enlargement of its extremity, presents a small wart-like and superficial prominence, the size of a millet-seed, on the posterior aspect of the last phalanx, and two commencing and superficial growths on its outer side near the thumb. On the posterior aspect of both thumbs are several small superficial bodies of a similar character. These growths are apparently painless and free from tenderness.

The lower extremities.—Both buttocks and the back of both thighs are affected in a manner similar to that which I have described as occurring in the scapular regions, and more or less symmetrically. There is the ecchymosed appearance, the same diffused nodular and undulating growth, but here the disease is more extensive and advanced at some parts. It presents itself here and there in the form of distinct tumours. It is advanced in both sciatic and popliteal regions, and in the neighbourhood of the left sciatic notch is a mass the size of a large walnut. On the anterior surface of the lower extremities there are soft and elastic masses in front of the ligamentum patellæ on either side, several small tumours over the patellæ and the front of the leg, and two or three slight periosteal enlargements on the front of the tibia.

The feet.—The disease in the feet is, to some extent, symmetrical. The last phalanx of the third and fourth toes of both feet is affected in a similar manner to the fingers, but the disease is advanced to the extent observed in the fingers only in the third toes. In these the parts are enlarged to four or six times their natural size. Extending outwards from the side of the matrix of the third toe-nail on the right foot is a horny growth the size of a small almond. The nails of the affected toes are furrowed as in the case of the fingers, those of the unaffected phalanges are also slightly affected in this way. There are a few small and scattered tumours, none larger than a pea, on both feet. They are similar in character to those on the legs; there is one small, warty-looking growth on the upper surface of the left fourth toe.

General condition.—The patient's general health is excellent. Her appetite is very good; her tongue is clean; her bowels are regular; her abdominal viscera are healthy; her thoracic viscera present nothing abnormal; her pulse is under 100, and the heart sounds healthy. The respiration 18 per minute, and the respiratory sounds are healthy. The lymphatic system does not appear to be affected by disease. The urine is very pale, sp. gr. 1010, but otherwise nothing abnormal is observed. The blood was examined, but presented nothing abnormal in its microscopic appearance. She is completely or almost deaf. When called by her name she gives no response, and does not behave as if she were aware of having been spoken to. Her attention is not aroused by loud noises immediately behind her. She is very intelligent, so much so, indeed, that the nurse, who also fulfils the duty of teacher at the hospital and is accustomed to form an opinion of the mental capacity of children, not suspecting deafness, believed for some time that the child's peculiarities were dependent on shyness and dull intellect. When the teacher directs her attention by signs and articulates the word "teacher" the patient imitates the word in a way, but it is not at all distinct. The nurse believes that any semblance of articulate speech which the patient possesses has been picked up by her from lip reading. Her father states that she has been heard to say "Tomma" for "Tommy," "Ditta" for "Dickey," and "Parvar" for "Father." My friend and colleague, Mr. Andrew Clark, who undertakes the duties of the aural department at the Middlesex Hospital, has been unable to detect any pathological lesion of the auditory apparatus. She struggled violently when an attempt was made to pass an Eustachian catheter. The external auditory canal and tympanum on both sides are normal. A watch placed against the temporal bone does not in any way seem to excite her curiosity. There is no evidence of paralysis or of affection of the senses of taste, sight, or touch, except in the last sense, as related, in the fingers and other affected parts of the body. There is no imperfection in her voice. She complains occasionally

of a little headache, but it does not appear to be of any duration. She does not in other respects suffer pain from her disease. She sleeps well.

Progress since admission.—I shall now proceed to give a very short outline of her progress since she came under observation at the Hospital for Sick Children. She was admitted into the Hospital for Sick Children, Great Ormond Street, on April 9th, 1872. She was placed under the care of Dr. West, and latterly of Mr. Thomas Smith, both of whom have courteously given me permission to use notes of her condition taken while the patient was under their observation. A description of her condition at that time may be made clear in a few words. Mr. Butlin, who was registrar at the time, took careful notes of the case, and I am thus enabled to refer to them and make her history fairly complete.

There were only three tumours on the forehead at that time. The condition of these, of the nose, and the ears, was not so advanced as now. The state of the gums was apparently the same; there were, however, several tumours of the scalp similar to those on the forehead, which do not now exist, but the cicatrices of which have been already alluded to. Those tumours are thus described by Mr. Butlin:—"Two tumours symmetrically placed on the back of the head, one on the right side, large, globular, and at first appearing to be full of fluid, for it appears to fluctuate; but upon closer examination it seems to be composed of soft solid, with a firmer base. The surface of the one on the left is ulcerated. A third tumour, also ulcerated, stands on the right side of the head, but has no equivalent on the left." He further states that the ill-developed hair covering these tumours was very scanty. "Above and behind each ear," he continues, "is a soft flattened tumour, the larger on the right side." The growth of warts on the back of the neck was much the same as now. There was almost no growth on the back. Below or about each knee the existing growth had commenced, and there was a small exostosis on either tibia. The disease of the fingers and toes has not

altered much; but some of the projecting nodules of the fingers which were in April "semi-transparent, with large and full vessels ramifying over them," have become partly shrivelled up and thickly covered with epidermis. The disease has materially advanced in the third toe of the right foot. On the 15th May the large tumour at the back of the head was removed by Mr. Thomas Smith. It bled freely. There was no capsule, nor was the tumour clearly defined in its boundaries. It was so closely connected with the skin on the one hand and with the pericranium on the other that it was with difficulty separated from these structures.

Chemical examination showed that the juice of the tumour was albuminous, and not mucous. The general character and microscopical appearances of the tumour were materially the same as of that described by Mr. Arnott further on.

When admitted under my care on August 9th, 1872, at the Convalescent Hospital, Highgate, the wound caused by the removal of the large tumour from the right side of the occiput had healed, the tumour to the left of the occiput had almost disappeared, but the ulcerated surface was still open, while the third tumour on the right side of the head had ulcerated and the scar had healed up.

Her condition was apparently in other respects materially the same as when admitted under the care of Dr. West.

The smallest tumour on the forehead was only noticed a week ago, while the others have increased steadily. The growths on the side of the nose and that on the ears have likewise continued to become more and more evident. The gums have gradually increased. On November 28th, after going to bed, she complained of headache, and on the following morning she appeared to feel sick, she refused her breakfast, but did not vomit; she was flushed, had headache, but no pain elsewhere. There were no new seats of disease then apparent. Her temperature had risen on the evening of her seizure to 99·4°, and it continued gradually to rise until the evening of the 30th, or the third day, when it

reached 101·6°. On this day induration and ecchymosis of the skin were observed over the seventh cervical vertebra and the left scapula. This new growth and most of that on the other shoulder, the buttocks, and thighs, appeared much about the same time, and have ever since grown rapidly. There has been a general tendency to increase in all the cutaneous and subcutaneous tumours except those on the neck. The growths on the chin and jaw were not observed until shortly after this period, but they may have previously escaped notice. From the 25th of December, 1872, until the 3rd of January, 1873, a second but less marked elevation of temperature occurred, the thermometer not rising above 99·6°. Nothing unusual was observed in her general health at this time, nor, although the disease was evidently spreading and increasing, on the legs and trunk were any special signs of inordinate increase noticed. Unusual changes may have occurred, however, without being observed, as I was out of town at the time and depended upon the testimony of a nurse, who is, however, a most intelligent and observant person. The general health has remained throughout remarkably good.

The temperature.—I have already said almost enough regarding the temperature. It has been, as a rule, normal. It has on several occasions, as on those related, continued slightly elevated for a few days, and about the time at which the extensive new formation at the back was observed, it reached as high as 101·6°. Whether the milder attacks of fever have had any connection with the commencement of new growth has not been clearly made out.

Six weeks ago my colleague Mr. Marsh removed at my request for microscopic examination a small tumour from the back of the ear about the size of a hazel-nut. A considerable amount of hæmorrhage occurred. The tumour was covered with a layer of skin. It was of a deep reddish colour, firm and elastic. Sections of this growth are on the table, prepared by my friend Mr. Henry Arnott, who has paid special attention to the study of tumours, and who has very kindly executed a few drawings, which afford

an excellent idea of the microscopic structure. He has also appended the following description of the character of the growth:

"Thin sections of the little tumour from the lobule of the ear, stained with carmine, present the following appearances:

"Viewed with a low power, magnifying about 40 diameters, the epithelial surface is seen to be perfectly normal, in no degree thickened, nor otherwise changed. The loose connective tissue supporting the *rete mucosum* is also in no respect different, either in form or in amount, from that usually met with in this position.

"Beneath this, however, and so slightly connected with it in all the sections examined as to leave a distinct space between the two, bridged over by only a few stray fibres here and there, is a growth of firmer consistence and more striking structure. The bulk of this firmer portion, constituting the new growth under investigation, is seen to consist of a homogeneous uniformly stained substance which pervades the mass in irregularly waving broad tracts, enclosing in their interstices smaller interlacing streaks of the same material and numerous small cells entangled in this finer meshwork the proportion of cells to the stroma tissue varying much in different parts of the same section. Examined with a $\frac{1}{4}$-inch objective, magnifying with low eyepiece 220 diameters, the elements of the new growth are seen to consist mainly of the homogeneous material already mentioned (which is everywhere rendered very distinct by being faintly [illegible] with carmine, the cells having always escaped the [illegible] spaces are for the most part [illegible] only a few small delicate [illegible] sections of blood-vess[illegible] much larger, of oval [illegible] small spherical or oval [illegible] the [illegible]

crowded into each enlarged space. When magnified 400 diameters many of the cells resemble still more closely those of cartilage, having a plump nucleus with nucleolus and a clearly defined double contour; some are imbedded singly in the hyaline substance, but the majority are smaller and clustered in the spaces. The high power shows many of these to have large oval nuclei and to be banded in tracts, as in growing fibrous tissue. None of the distinct waving parallel fibres of fully formed white fibrous tissue are to be seen. From these examinations the tumour would appear to belong to the connective tissue group, its histological elements being partly those of ordinary connective developing into fibrous tissue, and partly those of cartilage."

Treatment.—In addition to the surgical treatment, the patient has been kept on iodide of potassium in two-grain doses, but without any obvious effect one way or the other.

I shall now proceed to describe in a few words the condition of the other two patients.

Case 2.—Richard S—, 3 years and 9 months old. He appears to have passed through an attack of chicken-pox, but no other disease.

History of his illness.—The gums were first observed to be enlarged when he was about three months old, and the skin to be affected about six months afterwards.

Present condition.—The boy appears to enjoy excellent general health. He is apparently intelligent, but looks, and is, in fact, very sullen and stubborn. He scarcely ever attempts to speak, and has rarely been heard to articulate, except single words, as "mamma," "papa," "baby," "Anna," and "Dick." He was lately heard to say "No more trains," but never connected so many words together before, nor has he attempted to do so since. His voice is perfect; he phonates loudly and well. The gums are similarly, but to a greater extent, affected in this patient than in Ellen, rising in some cases much above the teeth. They are said occasionally to bleed, but moderate pressure

does not cause hæmorrhage. The tonsils are slightly enlarged; last month the left was deeply ulcerated, but the patient did not complain of this. A few of the deep cervical glands are slightly affected. There is a patch of thickened and glistening skin at the junction of the alæ nasi with the cheek, and a more extensive one on the eyelids at the inner canthus. The boy has got a bottle-nose, the lobe being swollen and hard. It presents slight bluish discolouration. There is a line, an inch in length, of warty growths, the size of a pin's head, on the right and posterior side of the neck. On the skin covering the back of the ear at its junction with the scalp is a long thickened patch of white and glistening fibrous skin, somewhat similar to the warty growths, and most marked on the right ear. The extremity of the middle finger of the right hand is very slightly enlarged and hard, and the nail is pushed backwards. There is a small warty growth on the dorsum of either finger. With these exceptions the boy presents no abnormality. There are no subcutaneous tumours. The general health is good, and the viscera are healthy. His temperature was taken regularly for about six weeks, but it presented nothing calling for remark.

Progress.—He has been under observation for nine months. During that time there has been slight increase of the disease of the eyelids and the neck. The commencing disease of the extremity of the middle finger is quite recent. It was not noticed until March 7th.

Case 3.—Anna S—, 2 years of age.

History of illness.—When two or three ı patch of affected skin, of a warty character, wı the back of the neck, and also swelling ı These have increased gradually, and the w growth has extended from the nape to the fron on both sides, and the skin behind the ear has ı affected. Within the last two months the sı junction of the left ala of the nose with the become the seat of growth.

Present condition.—The child, with the exception of slight rachitis, is healthy looking and cheerful, quite intelligent, and possessing intact all its senses. She already promises to talk well. The gums are affected in the same way and to as great an extent as in Ellen and Richard. On the skin in front of the right ear, and the cheek immediately anterior to this, is a small patch of isolated warty growth. There is a similar affection of the skin at the junction of the left ala of the nose with the cheek. At the nape of the neck, and in the mesial line, there is a circular patch the size of a shilling, of small, closely aggregated, warty looking elevations, from a quarter of a line to one line in breadth. They are of a shiny whiteness, and rather hard and fibrous to the touch. There are several lines of similar growth, extending anteriorly half round the neck from the spinal column. They are more or less continuous for distances of half an inch to two inches. The most developed, the largest, and probably the oldest in the group, are those nearest the spine. They are mostly isolated, but these small growths sometimes run into one another, the intervening skin being apparently healthy. The skin at the junction of the ear with the scalp is the seat of a continuous band of fibrous-looking growth similar to that on the neck. This child presents no other abnormal symptoms; there is no affection of the ends of the fingers, and there are no subcutaneous tumours. There is a chain of superficial, small, hard, but not tender glands on either side of the neck. There is no other glandular affection of the body. The general health is excellent, and the viscera are healthy. I have only seen this child twice, and cannot speak with any degree of minuteness or certainty regarding the progress of the disease in her.

Remarks.—I must, in the first place, apologise for the very tedious and lengthy character of my paper, and for having trespassed so freely on the indulgence of the Society. I thought it right, for the information of future inquirers, to enter with some degree of minuteness into a description of the conditions met with in the remarkable and unique

cases before you. With the continued permission of the Fellows I shall, in a very few words, refer to a few points of interest in the cases. I have not ventured to burden the confused and impossible nomenclature of dermatology by another name. Neither have I been able, with certainty, to determine the cause of the affection. Many of the features of the cases—the extensive cutaneous affection, and the connective tissue growths which form a prominent feature in the eldest child, and the appearance of those at an early period of life—lead one to place the disease in the group of molluscum fibrosum. Mr. Jonathan Hutchinson, who, as you are aware, is well entitled from his very large experience and his wide knowledge of the subject, to speak, has several times examined the patients, and says that he is tolerably certain that they ought to be placed in a family group with molluscum fibrosum; and other dermatologists, as Dr. Robert Liveing and Dr. Tilbury Fox, who have also examined these cases, have arrived at a similar conclusion.

There are, however, appearances, more especially the hypertrophy of the gums and the enlargement of the ends of the fingers in the two eldest children, which should not be lost sight of, which are of an unusually grave character, and which I believe have not been observed in molluscum. Again, the occurrence of this disease in more than one member of a family, if not unknown, has at most been met with only in a very mild form, while the condition of the gums hitherto not described in molluscum is common to three children of the same family. Moreover, the evident symmetrical character of the growth, and the mental and moral phenomena, slightly marked though they be, observable in one of the children, are more suggestive of nervous origin than we have been taught to expect in molluscum fibrosum.

I have alluded to the occasional elevation of temperature in the eldest child, lasting for a few days, and pointed out that this rise was coincident with a sensible advance of the disease of subcutaneous growth. Careful observations were

not made with the view of connecting the rise on the other occasions previously referred to with a fresh accession of the growth. The occurrence, however, of this apparent connection on the one occasion led me to suppose that the disease of the connective tissue might more particularly progress in an intermittent manner. I am confirmed in this by the condition of the nails, which present distinctly marked furrows, representing, most probably, repeated attacks in the fingers.

Before closing my paper I should like to say one word regarding the probable cause of the disease. There are, I think, two possible factors—either the bad hygienic condition under which the children were born and exposed after birth, or the parental consanguinity, or perhaps both.

It will be remembered that the eldest boy in the family, who was *born* under favorable hygienic conditions, is strong and healthy and entirely free from any abnormality of mind or body. The three younger members of the family, again, who were subjected *from birth* to the influences of damp for varying times, are all affected, and the disease is marked in them in a degree corresponding very much with the duration of their exposure. On the other hand, the disease still progresses in all the affected cases, removed although they have been for a long time from the influences in question; moreover, the eldest boy, who from infancy lived with the rest of the family, and was exposed for years to the same conditions as the patients, has nevertheless not shown any evidence of disease.

It is not improbable that the unsatisfactory state of their abode and probably increased poverty may have acted as an exciting cause of the remarkable changes in the patients. I am inclined to believe, however, that the more important and the predisposing element in the causation of the disease is to be found in the close blood-relationship of the parents. Such an opinion is, I think, more in consonance with our present knowledge. The facts at my disposal do not, I think, justify me in attempting to explain why an influence so strongly exhibited in the other members of the family

should have so strangely failed to express itself in the first born.

Postscript.—June 21st, 1873.—The growth on the back has become much less marked whilst taking three-grain doses of iodide of potassium; the disease elsewhere is not materially altered. She is now being educated at the Institution for the Oral Instruction of Deaf Children, Fitzroy Square. Mr. W. B. Dalby, aural surgeon to that institution, has kindly supplied me with the following note and opinion of her condition:—"External auditory meatus of natural formation and healthy. Tympanic membranes on both sides of natural translucency and curvature. Both Eustachian tubes pervious; vibrating sound passing through the conducting apparatus heard proportionately very much better than when communicated through the cranial bones. This, taken in conjunction with the absence of any history or appearances of disease in the outer or middle ear, point conclusively to the fact that the impaired hearing is due to a nervous lesion. It is quite open to question whether the child has ever heard well (although the father says that it did). If it has, the early age at which she became deaf and the evident progressive character of the affection show a similarity in some measure to what is seen in children who are the subjects of inherited syphilis. There is no syphilitic history to bear out this presumption."

Dr. Langdon Down pointed out during the discussion on this paper that in Ellen the facial development was much exaggerated, and that Richard presented the microcephalic type of cranium, while the ears were implanted very far back and obliquely. He believed that a scrofulous factor existed.

Since this paper was presented to the Society the mother has been delivered of another child. The baby presents none of the symptoms present in the others, and it enjoys good health, but there are numerous blotches (*nævi materni*) of a bluish colour, chiefly on the scalp and face.

DESCRIPTION OF PLATE VII.

FIG. 1.—Ellen S—. Case of molluscum fibrosum with hypertrophy of the gums; connective tissue tumours on the forehead and part of the scalp, &c.

FIG. 2.—Ditto. Showing enlargement of the last phalanx of the fingers.

Pl. VI

Fig. 1

Fig. 2

G. H. Ford

REPORT OF A CASE

OF

MOLLUSCUM FIBROSUM OR FIBROMA.

WITH OBSERVATIONS.

BY

GEORGE POLLOCK, F.R.C.S.,
SURGEON TO ST. GEORGE'S HOSPITAL.

Received March 8th—Read March 11th, 1873.

Mary Anne H—, æt. 33, a widow, was admitted into St. George's Hospital, November, 1872, under the care of Mr. Pollock, with tumours of the skin, such as are now usually known as "Molluscum Fibrosum," or "Fibroma," and consisting apparently of excessive hypertrophy or development of the connective tissue. These tumours vary in size and in shape to a very considerable extent; they are chiefly situated on the surface of the trunk, and are the growth of many years, having commenced in the early childhood of the patient.

The history of the case is a simple one. The patient stated that she had been the subject of these growths from her childhood—"as long as she can recollect." "She had never been free from them." They were small when she recalls to her recollection their early condition, and she had observed them slowly and steadily growing in size and

increasing in number. When quite young her mother once consulted a medical man relative to her child's condition, but since that time to the date of her admission into the hospital the patient never appears to have sought advice or assistance for the relief of her malady. It is difficult to give any exact description of the characteristics of this peculiar case, as no verbal account would accurately convey a right impression of its singular appearance. Photographs or drawings could alone illustrate its various conditions, and these have been secured and accompany this communication, while the patient herself has consented to be examined for the satisfaction of any one interested in the subject.

It will be observed on inspection of the patient,* or of the drawings and photographs, that the tumours occupy various positions (see Plate VIII), but chiefly, it may be said, that there are three large ones, and that all the rest, more than one hundred in number, vary in size from a small walnut to that of a split pea, or even something less. One large one is attached, at the back of the head, to the scalp and neck on the right side chiefly; in size about that of a small melon. When not supported, it hangs down to below a line drawn across the back from the spines of the scapulæ. Measured from its upper attachment to its lower free margin, it is about six inches in length, seven inches in breadth, and has a circumference of about twelve inches. The upper or posterior and outer surface is divided partially, and forms what may be termed two festoons or folds, the depth between these folds not extending more than about an inch into its substance. The base of this mass extends about an inch across the median line, a little below the occipital protuberance, to within about an inch and a half of the right ear. It is perfectly movable, painless on handling, and soft, with its surface covered partially by hair, and partially by thick coarse epidermis.

A second growth is to be observed on the top of the right shoulder. This mass does not project much; it is only slightly raised above the level of the surrounding skin, and

* Shown at the meeting.

its outline is distinctly marked by the especially dark colour and coarse condition of skin covering it. To a certain extent it is capable of being raised up from the deeper tissues, showing that there is an excessive development of connective tissue to permit of this freedom of movement. The measurement of this mass in a direction from the neck to the shoulder-joint is five and three quarter inches, and from before backwards in its greatest diameter three and a half inches; when raised up from its deeper attachment it can be made to project readily to the extent of two inches. In extent it occupies a space extending from three inches in front of the clavicle to the spine of the scapula. The most remarkable and the largest mass is in front. This grows from the right side of the neck, and extends in the shape of a long, thick, and broad pendulous flap of skin to below the level of the navel by an inch or two. This mass may be said to commence immediately behind the lobe of the right ear; its upper attachment or highest indication of commencement is narrow and somewhat pointed, and as the mass is traced downwards its base of attachment is seen to extend in breadth until it arrives at a line drawn across the front of the chest at a point some four inches below the upper edge of the sternum. Its breadth at this point of attachment is quite six inches. Its base of attachment in front may also be observed to take a somewhat irregularly oblique direction inwards, towards, and somewhat encroaching on, the base of the left mamma. It almost entirely covers the right mamma, lying over it, but not being attached to it, but the inferior attachment of the tumour is limited by the upper margin of the mamma. The weight of the tumour constantly pressing on the right mamma has caused the latter to project considerably to the right side, where it now remains, as may be observed when the tumour is raised.

The anterior surface of this large mass is very irregular, and is thrown into folds or festoons, which somewhat overlap each other and extend more or less from side to side, and increase in depth and width from above downwards.

The lower edge of the mass is quite free and more full and thicker than above, but rounded, soft, and moderately smooth. The tumour *in front* measured from its upper point to its lower free border eighteen inches; on its posterior surface, *i. e.* from its lowest point of attachment to its lower free border, eight inches. Posteriorly the skin is thinner and more inclined to be moist than in front, but in front between the folds or festoons it is also thinner than where exposed, apt to crack or excoriate, and secretes a thin serous discharge. This secretion or moisture has a peculiar and disagreeable smell. The folds on the anterior surface of the tumour, especially at its upper part, give it an appearance as if it consisted of convolutions, somewhat similar to the coils of intestine when massed together, and in a degree resemble an illustration in Dr. Warren's work on 'Tumours' of a tumour of the neck in a negress, to which further reference is made in a subsequent portion of this communication.

The general colour of the skin covering the larger tumour is darker than that of the rest of the body, its surface is also coarser and more rough; it is pretty uniformly marked by the obstructed orifices of sebaceous follicles, the contents of which may be readily pressed out to some length, and may be said to resemble small ascarides in size, and these obstructed follicles may be observed to pervade pretty generally the surfaces of most of the tumours, large and small. With respect to sensation, it is worthy of remark that over the larger portion of this large pendulous mass sensation is greatly impaired. A slight touch or the passage of the point of a steel pen lightly across its surface is not appreciated by the patient until a portion of the tumour near its attachment to the healthy skin is approached, but the more forcible handling of the mass, as lifting it up or pinching it, is readily detected at all points.

In addition to these three larger masses there are various other tumours occupying the trunk, from the size of a small walnut to that of a small pea. Some of these are quite solitary, some more clustered, some with a broad base,

others with constricted necks or pedunculated; some flattened and scarcely projecting from the surface, while others stand out and appear to be taking on the active stage of growth and tendency to become pendulous. Others have quite gained this condition, and offer what we may term tempting and convenient conditions for removal. One may be observed on the forehead, and a few on the arms and forearms. Some suspicious marks of fresh formations rather than the actual commencement of this morbid condition may also be observed on the forearms. One only is to be found on the upper portion of one thigh, otherwise the greater number and the best marked are to be found on the trunk, chiefly over the umbilicus, and mostly in front of the body. There is one slightly larger than the rest of these smaller ones on the right side of the neck in front of the upper attachment of the chief anterior tumour. It was from this that a large portion was removed some few weeks ago, and on the surface of what remains of it may be observed a cicatrix, the result of the operation. But it is important to remark that the mass now seen, and only some few weeks since the operation, has unquestionably increased in size. The wound of the skin requisite for the removal of the piece cut out was followed by a good deal of suppuration for so small and superficial an incision, and was tardy in healing. A second small tumour was snipped off the front of the body, but the wound readily dried up.

The woman has had two children, both of whom are healthy, and neither of them has shown the slightest signs of being affected in a similar manner to their mother. This poor woman was in a very reduced state of health on admission into the hospital, with a tendency to the constant occurrence of pustules on the face. She had undergone great mental distress and much physical hardship, and was in such an unfavorable condition that it would have been unwise, if not hazardous, to have attempted any operation for the removal of the larger mass at that time. Such, at least, appeared to be the case to a very recent period, and this opinion was confirmed by the results

of the very trivial operation for the removal of the small mass on the side of the neck. Operative interference with the larger mass has therefore been deferred, while the general condition of the patient has been attended to. She has of late so greatly improved that it is now hoped she may shortly undergo an operation with safety. This she is anxious for, that she may be relieved of the inconvenience occasioned by the size and weight of the tumour, and she is not only willing, but impatient, to have it over.

As regards the nature of these growths we may briefly state that it consists chiefly of hypertrophied connective tissue, with which, however, is mixed a rich cell growth. In order to authenticate or disprove this statement one of the very smallest of the tumours was snipped off, and also a large portion of that already alluded to. Both these specimens were submitted to microscopic examination by my friend and colleague Dr. Whipham, to whom I am indebted for the following most complete report as to their structure:

"Two portions were submitted to microscopic examination, viz. 1, a small sessile tumour, which Mr. Pollock removed entire; 2, a piece of a larger growth.

"1. The small tumour was oval, lobulated, and of about the size of a currant. Though sessile, it was in some degree smaller towards its base than at its apex.

"It was entirely covered by layers of epidermis and rete mucosum, which in no respects differed from those structures as found in healthy skin.

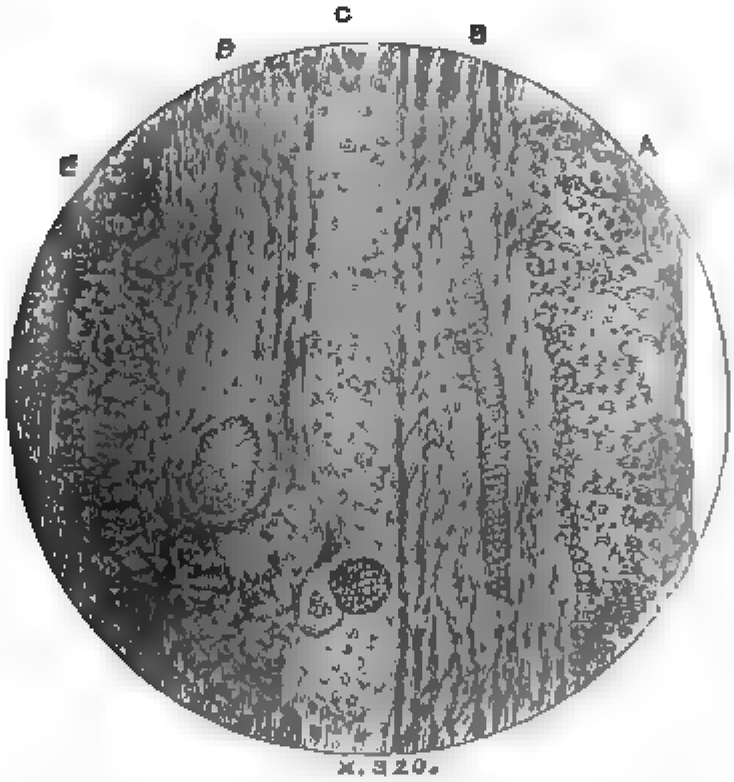
"Immediately beneath, and closely connected with the rete mucosum, was a layer of wavy well-defined fibrous, mixed with a small amount of yellow elastic, tissue, which in many places exceeded in thickness that of the epidermis and rete together. This fibrous layer absorbed the colour of carmine more readily than any other part of the tumour. Proceeding inwards, *i. e.* towards the centre of the growth, the fibrous tissue, which constituted its bulk, became split up into separate wavy bands, varying considerably in thickness;

and between these bands was an abundant growth of small round or oval cells, which were closely aggregated in large groups or arranged in lines between delicate strips of fibrous tissue. Occasionally this cell-growth lay immediately beneath the rete, and in such places the carmine-tinted fibrous layer was absent. In the central parts of the tumour, fibrous, mixed with yellow elastic, tissue abounded; the fibrous tissue was less dense and more wavy; it was split up into more distinct bands, separated from one another by wider interspaces, which interspaces were either empty or were occupied by the cell growth above mentioned. The tumour was well supplied by blood-vessels, whose fibrous tunic was rather thick and dense when compared with the size of the vessels. The vascular supply was more abundant in the central parts of the growth.

"2. In the portion of the larger tumour the microscopic appearances were in most respects similar. But in this growth sebaceous and sweat-glands were present, but scarcely so numerous as in healthy skin. These organs were in *all respects* natural. Occasionally, though rarely, a hair was found, always presenting a healthy appearance.

"The growth, therefore, is due partly to an excessive hypertrophy of the connective tissue of the true skin, and partly to an abundant cell growth occupying interspaces between the bands of fibrous and elastic tissue, which, as has been shown, composed the chief part of the growth. Neither the large pendulous nor the smaller sessile tumours depend upon any alteration in the epidermis, rete mucosum, glands, or hair-bulbs, as far as can be made out. In all probability the disease is a case of 'molluscum,' while at the same time some parts of these tumours bear a certain resemblance to a form of disease described by later writers under the name of 'dermatolysis.'" A drawing of the microscopic appearances by Dr. Whipham is appended.

This disease, though to some extent a rare one, is by no means unknown to surgeons, nor have its characteristics been overlooked by pathologists. Virchow has published



A. Epidermis and rete mucosum.
B. Perfectly formed fibrous tissue, with no vessel in section.
C. Cells arranged in a more or less linear manner.
D. Fibrous layer. An artery in transverse section.
E. Dense fibrous and yellow elastic tissue. The section rather opaque at this spot.

an engraving of a very remarkable case.* In order to give the members of the Society an opportunity of comparing it with the case already described and another to be presently mentioned, copies of the engraving have been made for me by Messrs. Maull in photography. The engraving (which was shown at the meeting) represents the appearance "of a woman, æt. 47, who had over the whole body numerous small growths, and one very large, which had slowly developed themselves during some years. Some were very minute, and others larger. The largest was fixed or held by a broad base over the left lower ribs, and extended from the linea alba to the spine, and hung down over the hip. On its surface were many small secondary tumours, the whole covered with thin smooth skin. It felt soft and almost fluctuating. When removed it weighed 32½ pounds.

* 'Die Krankhaften Geschwülste,' i, 325.

It was juicy, and an albuminous fluid was easily pressed out. It was divided by septa, from which passed a finer network. The smaller tumours were under the superficial skin, projecting into the rete Malpighii. The microscope showed a soft tissue rich in cells surrounding an actively growing granulation-tissue."

A case somewhat similar to this is mentioned in the catalogue of the museum of Guy's Hospital, the models of which in wax are to be seen there. These models were taken after death, by Mr. Towne, from a man over eighty years of age. There was a large tumour, the size of a melon, on the back of the head; more than a hundred of various smaller sizes on the body, front and back; and one large one hanging from the nates, some 16 lbs. in weight. The body and limbs were equally affected. The tumour on the back of the head on one occasion was injured, and bled profusely; but considering the age at which this man died, these growths cannot be said to have materially shortened life in this instance.*

A very remarkable case is reported in the 'Transactions of the Pathological Society' (vol. xvi) by Dr. Henry G. Wright, and is especially interesting from its great resemblance, in its chief features, to the case of Mary Anne H—. I have therefore secured photographs of the engravings for the satisfaction of those interested in this subject, and am induced to relate shortly a few particulars of the condition of this patient. "A woman, æt. 34, single, was admitted into the West Riding County Asylum, 1864. She had large pendulous growths of skin on right half of neck and chin, and partially over the middle of the sternum, extending about as low as the umbilicus; and also behind, over the cervical and upper three or four dorsal vertebræ. In front the attachments extended along the median line, from chin to sternum, as low down as a line drawn across from nipple to nipple. Behind, along the median line, over the whole of the spines of the cervical and three

* See No. 168, Models of Diseases of Skin, in 'Guy's Hospital Museum Catalogue.'

or four upper dorsal vertebræ.* They cover the right side of the neck, extending a little above the right ear. They also involve the right half of the chin, and extend across the median line towards the left side. There is a smaller growth on the upper part of the right shoulder, which on admission was ulcerated, but healed, and had grown since from half to three quarters of an inch. The folds of skin hang down as low as the umbilicus in front. The growths were first noticed at fourteen years of age, and had gradually extended and increased in size since then. There was some enlargement or growth in the scalp above the right ear.† Dr. John C. Warren, in his chapter on dermoid tumours, describes a case which probably partook of the nature of those already described, and to which he applied the term "eiloides" from its coil-like character. He remarks that "this is a true production of the cutis, and continues so throughout, although it may become very large." "If removed," he adds, "it is reproduced, and ultimately affects the patient's health."‡

In presenting these observations to the Society I feel I have nothing new to offer or suggest respecting the characters or treatment of this interesting disease. It occurred to me that as the specimen now produced was so remarkable in its features, and in its occurrence so rare, it would not have been right to allow the members of the Society to lose the opportunity of examining it prior to the removal of the larger mass. At the same time it appeared to me that by collecting photographs of a few more well-marked specimens from authentic engravings much additional advantage would be derived from this communication, and the illustrations would always be available for reference hereafter.

It is, perhaps, worthy of remark that there is to a certain extent a correspondence between the positions of the larger tumours found on the patients whose cases have been mentioned. In two of them a large tumour grew from the

* See 'Path. Trans.,' vol. xvi, woodcuts 23 and 24.

† 'Transactions of Pathological Society,' vol. xvi, p. 269, 1865.

‡ Warren, 'On Tumours.'

back of the head; in two the largest growth was from the right side of the neck; a large tumour hung down from the side and nates in two others. Secondary and smaller tumours appear to have been situated at any part and over all parts of the body, but in two the legs were quite free, and in one case there is no mention of any tumour on the arms. The favorite locality, from all I have been able to ascertain, is the trunk; the arms and legs appear often to be free.

With respect to treatment I can say but little. I propose to remove the large mass in front of the chest in the course of a few days, and in doing so shall take especial precautions to guard against undue loss of blood. I cannot help entertaining a feeling that the relief from the operation may only be partial and temporary as far as regards this one individual tumour, for from the circumstance that the smaller one on the neck has grown since the greater portion was removed I am quite prepared to find that recurrent growth of the large pendulous flap may take place after the proposed operation. In the case related by Dr. Warren the tumour was removed from the neck, but recurred in about eighteen months. But, notwithstanding such a prospect, with the urgent wish of the patient, and considering the great local inconvenience of the tumour, it must be acknowledged that the proposal to relieve the patient by operation is perfectly justifiable.

Postscript, Aug. 8th, 1873.—The large mass was removed on the 15th of March. The flap was held up, and a superficial incision made along the border of its lower attachment, merely through healthy skin. I avoided cutting through any portion of diseased skin, and so did not carry the incision to the anterior aspect of the flap. Double ligatures were then passed, about an inch apart, through the base of the tumour, from the incision behind to and through the anterior surface. The ligatures were tied so as to secure and compress as much as possible every vessel at the base. The flap was then removed by a sweep of the knife. Notwith-

standing the precautions taken, some few small arteries had to be tied separately, but the mass removed gave evidence of the advantage of the means taken to prevent loss of blood, for from it escaped a large quantity, both venous and arterial. Had not the ligatures been applied before the removal of the mass, it was evident very considerable, and very troublesome hæmorrhage would have resulted, whereas very little more blood was lost than that contained in the tumour. The patient did well after the operation; and was pleased at the relief afforded. When seen some four months afterwards she was quite well, with no indication of regrowth at the part operated on.

The mass removed weighed 2 lb. 6 oz. It had a broad base which measured 11 in. × 3 in. The surface was corrugated and the skin thickened. On the cut surface of the base may be seen the openings of numerous large veins; and injection shows the tumour to be extremely vascular. The internal structure of the tumour resembles that of condensed and hypertrophied cellular tissue.

DESCRIPTION OF PLATE VIII.

Mary Anne H—. Case of molluscum fibrosum.

FIG. 1.—Front view.
FIG. 2.—Back view.

FIG 1

FIG. 2

AUTOTYPE LONDON

THE PATHOLOGY OF LEPROSY;

WITH A NOTE ON

THE SEGREGATION OF LEPERS IN INDIA.

BY

H. V. CARTER, M.D.,
BOMBAY ARMY.

COMMUNICATED BY

E. SYMES THOMPSON, M.D., *Hon. Sec.*

Received February 28th—Read March 25th, 1873.

THE following remarks are the result of observations made in Western India, 1860-71.

I. *The pathology of leprosy.*

1. *Summary of diagnostic and anatomical characters* :— 1. Alterations in the *skin*.—These are either (*a*) produced by the so-called "tubercles," whose appearance is well known in Europe; or (*b*) such as are now frequent in the East, and characterised by atrophy rather than by swelling; these latter forms of eruption are usually associated with impaired nerve influence; inclusively, they are widely known in Western Asia under the term "bărăs," and the most typical of them corresponds to the "leuke" of classical

Greek writers. In certain characters bărạs may, I think, be compared with the "lepra" of the present day (Willan), only that the "scaly" feature is wanting, because in warm climates conditions are not favorable to the accumulation of dried epithelium on the skin, and it might be termed "*lepra leprosa*" in English nosology.

2. Destruction of the *cutaneous nerves,* this is the characteristic lesion of the disease.

3. Hence result the marks presented by lepers—large pale or dark blotches on the skin; anæsthesia of the extremities and trunk, with subsequent wasting, distortion or partial loss of the hands and feet, the muscular power, however, remaining more or less intact.

If "tubercles" predominate there ensue a well-known disfigurement of the visage, and scattered swellings in the integument, as well as on the tongue and palate, and in the larynx; commonly, anæsthesia and pale patches coexist with these "tubercles."

Subsequent changes are slow in progress, and are indicative of malnutrition of the body, both local and general, and hence susceptibility to corresponding hurtful influences.

The only infallible signs of leprosy, in my opinion, are one or other of the above marks. I know of no truly characteristic prodromata.

It will be observed that no reference is here made to symptoms of *visceral lesion;* indeed, there appear to be none peculiar, and while it is true that lepers die bearing the mark of their disease upon them, this event does not take place at any one period of the malady, or at any particular age of the patient, or in any uniform manner; in short, death occurs from simple exhaustion or from chronic dysentery or diarrhœa, or other intercurrent affection, induced by the want and exposure which are too often the lot of the miserable leper.

4. *Morbid anatomy and histology.*—The structural changes observed are due to exudation or deposit in the skin and nerve-trunks of a firm, translucent, colourless, or pale reddish material, which may be distinguished

by the borrowed terms "hyalin fibroid" and "hyalin granular."

As regards the skin, conjunctiva, and adjoining mucous membrane of the mouth, this deposit (here hyalin granular) first appears within or immediately beneath the cutis or mucous membrane; the hairs and glands are more or less involved, the functions of the nerves being impaired; those papillæ containing tactile corpuscles are early affected, and I have also found the pale muscular bundles with their nuclei in a state of fatty degeneration. The blood-vessels are not particularly altered, and ulceration of the "tubercles" is not common, except in the mouth and larynx.

As regards the nerves, this deposit (hyalin fibroid) first appears between the individual nerve-tubules and within their common envelope, *i. e.* the neurilemma of the funiculus; the outer sheath of connective tissue is hardly changed. By accumulation of the new material the nerve-tubes are separated, compressed, emptied, and eventually destroyed.

The microscopic characters of this leprous deposit favour the view of its being an exudation capable of a low grade of development. It is true that it first appears in localities occupied by connective tissue; this is certainly the case in the skin, and probably so in the nerves, for, although I found it hard to say whether or not any intertubular substance normally exists in ordinary nerve-bundles, yet their delicate neurilemma and the primitive sheaths contain cells or nuclei, whence proliferation might start, and I have sometimes found the new material cellular rather than fibroid in character. Still, there is an abundant neoplasm whose source appears exudative. At a subsequent stage degeneration, fatty or even calcareous, may take place.

There are some minute differences in the appearance of the deposit as it is found respectively in the skin and nerves, which need not now be insisted on.* (See Plate IX.)

* Full details of the symptoms and morbid anatomy of leprosy are contained in my article published in the 'Transactions of the Bombay Medical and Physical Society,' new series, vol. viii, 1862.

In sixteen autopsies, carefully performed, no trace was noticed of deposit in the muscles, bones, or in any of the viscera; the brain and spinal cord were wholly free from deposit.

I should add that the inguinal glands (superficial) may be enlarged, the nipples may become nodular in both sexes, and I have seen the testes both enlarged and wasted; yet not one of these circumstances, when thoroughly sifted, appeared to really impugn the statement that the leprous disease is, in its manifestations, limited to the cutaneous system.

5. *General characters and prevalence.*—True leprosy prevails in all parts of the Bombay Presidency, though commonest of all on the sea coast at about 18° N. lat. (1 leper in 430 inhabitants), yet it is almost as frequent (1 in 550) in an inland district elevated 2000 feet, with no large rivers running through it; in short, the disease is found under all climatic conditions, whether of soil, vegetation, temperature, or rain-fall; its intensity has no ascertained relation to the sub-varieties of climate in Western India, and if it have to geographical conditions, this is only because they are connected with racial distribution.*

The disease affects all classes of the community, varying in proportions roughly estimated at 1 leper in 400 of population (Kolis and Mahrathas), to 1 in 2000 (Parsees); also both sexes, but males as 4·38 to females 1; it commences at all ages, but oftenest at 20—40 years in men, or somewhat earlier in women; it does not greatly shorten life. It spares no rank, and is not regulated in frequency by affluence or penury, and, in short, on wider review, it appears to prevail independently of such influences as social status, habit, customs, or diet.

The distribution of leprosy in Western India is, however, I opine, connected with that of the races of men belonging

* Full details of the characters and prevalence of leprosy in the Bombay Presidency, based on official returns, will be found in the 'Transactions of the Bombay Medical and Physical Society,' new series, vol. xi, 1871.

to the population. Thus, the aborigines and their existing descendants (including Mahrathas) seem more infested by it than are the higher castes (or Aryans); later invaders, as Arabs, Africans, and Jews, have brought the disease with them. The Portuguese half-castes are affected in full proportion.

One prime feature of the disease claims special notice, namely, its capacity of transmission in a latent form (the so-called innate predisposition) from either parent to their offspring. Upwards of one fifth of all lepers (8220) actually acknowledge such origin of their disease; thus, of 1564 instances, heredity in the direct line was named in 64·2 per cent. (the father being mentioned by 43·8 per cent.); through the collateral line (uncle, &c.) in 14·5 per cent., or as often as through the mother; while 21·3 per cent. had brothers or sisters affected. "Atavism" or reversion is not unfrequent.

Lastly, since, through the universal and long-prevalent influence of caste distinctions, the people of India are subdivided into sections, large and small, between whom no intermarriage is permitted, there has resulted a state of kinship, however at times remote, which cannot but have had, in conjunction with force of heritage, very decided influence in maintaining or even extending among them a leprous taint; and it is, I think, to the combination of racial proclivity, heredity, and concentration through caste restrictions, that the source, long persistence, and general prevalence of leprosy in Western India should be attributed.

6. *Seat, nature, and causes of the leprous disease.*—The following conclusions seem deducible from collation of orderly arranged data.

1. *Seat of leprosy.*—The defect constituting this disease involves the constitution, inasmuch as it is transmissible in a latent form to offspring; but with respect to its outward manifestation it is restricted to certain tissues of the integument or cutaneous system.

Evidence to this effect is both negative and positive, and may thus be summarised:—First, in sixteen unselected autopsies no uniform morbid change was observed in any of the great cavities or their contents; the blood itself, so far as examined, varied in appearance, and the incidental character of the cause of death was sufficiently apparent. I have lately reconsidered the point, and am still of opinion that there is at present no positive evidence that leprosy, even in its worst form, involves the deeper-seated structures of the body.

Again, it is not every tissue of the integument which is directly implicated, but, as before stated, first the nerves, next the cutis; and the nerve-affection I hold to be pre-eminently the more characteristic. Thus, both "tubercles" and "eruption" are invariably associated with nerve lesion, while this latter may, for a time at least, exist alone. By repeated dissections I have ascertained that so many of the cutaneous nerves are actually diseased as to render the assertion credible that none are not liable to become affected.

Very noteworthy, too, is the fact that these nerves—such as the branches of the fifth pair on the face, of the cervical plexus to the ear, &c., of the brachial plexus and intercostal to the upper extremity, of the dorsal nerves, and of the lumbar and sacral plexuses to the lower extremities—are all diseased beyond that spot in the deep fascia of the muscles at which they become strictly cutaneous, or, in other words, only in that part of their course in more immediate relation with the skin, and they are seldom altered except by subsequent wasting in the deep-seated part of their course. I never detected any constriction at the perforation in the fascia which could mechanically account for this marked limitation of disease, so significant in its meaning.

But sooner or later the deeper-seated main nerve-trunks of compound function become more or less diseased, and at first sight this might seem an objection to the view propounded. Let it, however, be noted that the nerves, *e.g.* the

ulnar at the elbow and wrist, the median at the wrist, the popliteal in the ham, the posterior tibial at the ankle—are earliest and most, if not alone, affected where in their course they approach nearest to the skin, and where, one might hence almost infer, some irritative influence reached them. It is true that the spots I have mentioned being near to joints, the nerves are there exposed to repeated traction during movements of the limbs, and that such traction, entailing pressure, may be sufficiently irritative to induce leprous deposit; but in many cases it will be found that there is a direct continuity between the affected main trunk and its altered cutaneous branches, which indicates extension of disease from the latter to the former. It should be specially noted that not every part of the larger trunks in these instances is impaired, since motor paralysis is rarely present to any extent, even in the worst cases of leprosy; and the inference is therefore plain, namely, that it is the sensory element of these compound nerves which alone is implicated.

I would here remark that, in my estimation, "trophic" or nutritive nerve-tubules are commonly associated with those termed "sensory;" and I will add equally explicitly my opinion that the process of nutrition may be directly affected by nerve-influence: the grounds of these statements are recorded elsewhere.

It may be urged that in the so-called "tubercular" leprosy the nerve affection is of very subordinate importance. Certainly it would seem as if the force of the disease, then I believe at its greatest, were in this case expended on the skin; yet without pressing mere argument I must observe that even in tubercular leprosy (which is comparatively rare in Western India and adjoining countries), the ordinary nerve-disease is well marked, as is proved by the dissection recorded in my first paper, as well as by those of the fatal cases of mixed tubercular and anæsthetic forms of the disease.

I have, too, been impressed by the fact that the seat of the tubercles on the face is just where the cutaneous branches

of the fifth pair emerge and pass onwards; notable is this with reference to the first division, or supra-orbital nerve.

Again, I have so repeatedly found association of tubercular enlargement of the ear and disease of the great auricular branch of the cervical plexus, that I should be disposed to make this instance a stand-point for discussion; the question being whether the phenomena are cotemporary or consecutive. Without prejudging, I think there are good grounds for believing in the priority and even causative precedence of the nerve-affection; nor is there any error in assuming that impaired nerve-influence might dispose to leper deposit in the skin.

As respects the characteristic eruption known as "Baras," there are reasons equally or even more forcible for connecting it with the coexisting nerve-disease, but their repetition in this place is not needed.

Thus the characteristic nerve-lesion of leprosy appears amply competent to explain all the more serious symptoms of the disease—the loss of feeling, shrinking, drying and coldness of the skin; the wasting and final disappearance of the digits, or even next segments, of the extremities. There is, indeed, such a close correspondence between the areas thus changed, and those of the nerves appertaining, that, having regard to the apparent sequence of symptoms, but one inference seems possible, to the exclusion of the idea that the ordinary tissues and the nerves are affected by a common change.

Finally, it should always be remembered that the effects of locally suspended nerve-influence greatly depend upon attendant circumstances. In India lepers belong for the most part to people of primitive habits and limited means; they are therefore commonly without the appliances adapted to mitigate their complaint; and what is worse, scores of them are yearly expelled their homes, or sent forth as wandering mendicants, being subject to such want and exposure as would be detrimental to even healthy people; and hence most of their sufferings. On the other hand, I have known instances where with comforts and due care

the worst degrees of the disease have been so mitigated that life was prolonged for years and but little physical pain experienced.

II. *Nature of the disease.*

The proximate cause of leprosy appears to consist in a faulty condition of certain tissues of the integument—a defect, perhaps, of development, certainly a quality transmissible to offspring.

The existence of a dyscrasia or primary blood change seems hypothetical and even needless.

The nearest approach to evidence of such dyscrasia which I have witnessed in India is the stunted growth with arrested development of some young lepers, the victims of the tubercular or mixed tubercular and anæsthetic forms of the disease; but as these individuals were born and reared in dire poverty, and had been subjected since the outbreak of their complaint to additional privation and exposure, there already was apparent sufficient explanation of their miserable condition. In such cases, I may add, it is usual to see ulceration both of the digits and of the "tubercles" in various parts, especially in the mouth and larynx; internal ulceration and subsidence of the nose may also coexist, which I have thought traceable to disease of the nasal branch of the first division of the fifth cranial nerve, and direct and more striking illustrations of similar correlations are recorded in my dissections.

Occasionally the urine is albuminous, with other signs of degeneration of the kidneys, but since I have not found in the renal organs a leprous deposit, these instances strictly have the force of coincidence only, and are not evidence of a dyscrasia peculiar to the disease under notice.

In the absence of more positive data, reasonings from analogy, *e.g.*, of leprosy with syphilis, strumous affections, &c., need not be entered on here; it may, however, be granted that conclusions negatively founded can be but provisional.

III. *Etiology.*

Of the numerous so-called causes of Leprosy the following alone need be noticed here: an inherited predisposition; contagion and inoculation; endemic influences.

Briefly, and with respect to experience in India, I am of opinion that heredity is the common cause of the complaint. This subject has been already referred to, but as more particularly noteworthy should be considered the fact that the inherited disease is identical with that not known to be derived; and it is from no desire of resorting to the obscurity yet enveloping the subject of heritage in disease that I am disposed to hold this fact as supreme.

Respecting contagion or inoculation, the evidence of crucial test is necessarily wanting; hence opinions will vary according to degrees of accuracy in available information, and according to the felt cogency of argument. In my Report based upon returns of all known lepers in the Bombay Presidency, the instances of married couples who are lepers are discussed, and it is shown that no sound inference can be based upon them; it is also mentioned that while sixty-six widows had leper husbands, only eighteen widowers had leper wives; but here again the circumstances are such that no inference is sure. The rare cases in which the disease might be traced to possible contagion or inoculation were too few to allow of a general affirmative statement on this subject.

With regard to endemic influences the data from a single sphere of inquiry may be deemed defective; yet I know of no one feature of air, soil, or water which has certainly led to this disease. Europeans, both civil and military, inhabiting every part of Western India, often for long periods, are virtually never affected with leprosy. Exceptional instances are, indeed, so rare that, since liability to contagion is usually denied, one might resort to the supposition of latent taint.

The correlated prevalence of malaria with leprosy, not to

mention closer relationship, has not been established, and the same must be said of local peculiarities in diet, habits, and hygiene.

Admitting, however, the frequent presence of an innate predisposition to the disease, its development may be favoured by all causes leading to deterioration of health, such as an unhealthy climate, bad or insufficient food, neglected sanitation, social hardships and the like; whence the numerous agencies to which leprosy has been attributed as effect to a cause. The converse also holds good; lepers do seem to improve on change of climate, and it has been assumed that the disappearance of leprosy in Europe may be directly referred to improvement in the diet and general condition of the people; but on taking a wide review none of the external influences now named can be accepted as true causes in originating, or, conversely, in eradicating the disease.

Lastly, no evidence is forthcoming that in India, at least, a natural tendency of the disease to subside exists.

NOTE

ON THE

Segregation of Lepers in India.

By a nominal return, made in 1867, of all known lepers in British territory under the Bombay Presidency, it appears that in 10 only of the 16 districts, and exclusive of Sind, their number is upwards of 8000; in four districts, placed in juxtaposition, the names of 5309 lepers were registered—a number equal to 1 affected in 600 of the population, and in one or two of them the number of lepers was nearly as great as in the whole of Norway, according to a former estimate.

When the results of the recent general census of the Presidency are made known, more complete data will be at hand; and in the future it may be possible to ascertain whether or not leprosy is on the increase—a question to be answered affirmatively in certain British Colonies, and in Norway—but meanwhile sufficient is known to engage attention with a view to remedial measures.

It was recently affirmed by high authority that as a means of checking the progress of leprosy, the compulsory segregation of lepers was not to be recommended; and, *inter alia*, it was then advanced that the decline of leprosy in Europe is attributable chiefly to improvement in diet, as well as of general hygiene, identical results being predicated in India on similar grounds. Apparently the assumption that leprosy is not contagious led to this discountenance of leper asylums, and the necessity for consistently explaining the extinction of the disease in Europe during the middle ages probably led to this result being referred to improved hygiene.

Yet neither of these considerations is final. For although the view that leprosy can be propagated by contact may not be upheld by the majority of medical men in India, whose opinions are necessarily based on ordinary observation, yet all would probably admit that special and precise information is needed to thoroughly settle the question with respect to all stages and forms of the disease; facts, too, are slowly accumulating in other parts of the world which at least render plausible the assertion that the casual inoculation of leprous matter is one actual means of spreading the complaint. But on other grounds the segregation of lepers seems most desirable, for in India, as long as diseased people are allowed to freely intermix with others, this scourge will be propagated by marriage and by intermarriage of the affected, and the morally bad effects on the people of permitting them to harbour in their midst miserable and often disgusting cripples are, whenever the number of such subjects is considerable, quite undeniable. Custom, among the middle classes, does not demand more than the lodg-

ment of these wretched objects apart in a pent-house or hut erected in the outskirts of the village, while the poorer classes expel their lepers of both sexes, thus adding to the army of mendicants, and otherwise diffusing much evil. On the grounds, therefore, both of humanity and of expediency does it appear desirable that leper asylums should be made recognised institutions, adapted to meet a wholly special need of the country.

Respecting the statement that improved hygiene has in some countries and in past ages led to the extinction of leprosy, the reasoning seems to be defective, for all civilised nations have, and do yet, put in force measures more or less strict of quarantine against lepers, and this with no dissent on the part of the community; and as regards the decline of leprosy in Europe till its extinction at the close of the fifteenth century, after a prevalence of 400 years, dating from its supposed introduction by Crusaders returning from the East, the most patent fact seems to be not that of a general improvement during this period in the diet and habits of the people—to which change, indeed, due influence must always be allowed—but rather that of the rigorous measures adopted for checking the progress of this new scourge. Thus, "Laws were enacted by almost all the princes and courts of Europe to arrest its diffusion amongst their subjects; the pope issued bulls with regard to the ecclesiastical separation and rights of the infected; a particular order of knighthood was instituted to watch over the sick, and leper hospitals or lazar-houses were everywhere instituted to receive the victims of the disease." During the middle ages almost every large town in Great Britain had a leper hospital or village near it for the reception and separation of the diseased, and some cities were furnished with more than one. These hospitals were intended for the isolation of the infected, not for their cure; they were charitable and religious rather than medical institutions.

"In Great Britain, as on the Continent, those affected or supposed to be affected with leprosy were obliged to seclude

themselves from society or enter a leper hospital, both by general custom and usage and by direct legal enactments." . . . . It is not necessary to quote further from Simpson's account of leprosy; and without pressing mere argument, it cannot but be admitted that the utter ban under which lepers were put by law and custom had the greatest influence in checking and eventually eradicating their intolerable malady; it may be said that it was thus "stamped out."

Nowhere in the east does there appear ever to have existed such a machinery; and there the disease is probably as rife as ever, and certainly rendered more inveterate by this tolerance for centuries.

To mention the old harsh leper laws except with deprecation might not be acceptable; yet if the alternative to their partial imitation in India be the hope "that a marked change in the habits of the native population will ensue upon the increase of divers industries, the improved cultivation of the land, the less frequent recurrence of famines, and the consequent amelioration of their general condition from year to year, and that better food, better clothing, and better housing, with greater personal cleanliness, will lead to the abatement of leprosy, may be confidently anticipated," there can then be no such early prospect of this scourge being eradicated as might consistently be anticipated from unsparing employment of all the means which experience, both past and present, would indicate as the more efficient.

Assuming that practical measures are positively needed for the purposes of checking the progress of disease, relieving burdened poor, and removing from sight a wretched class of mendicants, three measures at least are open to adoption:—1. Strict segregation of all indigent lepers (as a preliminary measure) in fit houses or villages within the district to which they belong. 2nd. Disportation of the affected to a central, cool, and healthy locality. 3rd. The establishment of hospitals for the accommodation of lepers alone, should any plan of treatment be found to be strictly curative.

APPENDIX

TO THE

Preceding observations on the Pathology of Leprosy.

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1. It seems fit that reference be made to the views of Professor Virchow on this subject. These I have quite recently perused with much interest in the French translation of Virchow's work on the 'Pathology of Tumours,' vol. ii, p. 486 *et seq.*, 1869.

2. A chief point—namely, that of the constitutional nature of leprosy—Virchow regards as yet undetermined, although venturing on the statement that he is perfectly convinced that tumours of this kind (*i. e.* leprous tubercles) are to be met with in deeper-seated parts and giving, as evidence, the instance of a testis found to be the seat of such tubercles.

Now I, too, have found the testis enlarged in a diseased leper, but then the change was referrible to syphilitic disease. Nor can the implication of the superficial lymphatic glands, or the sub-mucous tissue of the tongue, palate, and larynx be regarded as different or better evidence of constitutional taint than the changes in the skin itself, and in the cutaneous nerves, which I am disposed to insist upon more than other previous writers, so far as my acquaintance with them extends.

Lastly, while it is true that the negative testimony afforded by the Bombay dissections can necessarily have but a certain value, yet in my opinion it is quite premature to decide that the deeper-seated organs are affected in leprosy, and this conviction is not shaken by the scanty counter-evidence hitherto adduced. Be it remarked that the appearances depicted in the oft-quoted 'Monograph' of

Danielssen and Boeck are not now insisted upon as characteristic of visceral lesion peculiar to leprosy; on the whole, therefore, this disease resembles lupus rather than syphilis, and it is sufficient to concede that a constitutional form of leprosy may be by-and-by made out, as has been done with respect to syphilis, there being at present neither sign nor need of such revelation.

3. Believing that the facts accumulated in India are more numerous and of wider range than others before discussed, I have sought to give expression to the plain inferences derived from them. As regards the causes of leprosy, one must regard it as unproven that they are certainly either endemic and local climatic influences or dietetic faults. And respecting the supposition of Professor Virchow that there must be a special cause of leprosy, considering that the disease has now disappeared in the most complete manner from parts where it once prevailed, all that need be said is that the circumstance of its well-nigh total disappearance in Europe is well and fully explained (such is the view here advanced) by the utter exclusion from mixture with their fellows, to which lepers were subjected by law and custom, whence it became impossible that a tainted progeny should persist in the land. Even then the disease was not extinct until after the lapse of four or five centuries at the shortest; and it has been well remarked that the last lingering traces of leprosy in a once infected locality will be found in groups of individuals connected together by family ties.

DESCRIPTION OF PLATE IX.

Microscopic Character of the Morbid Deposit in Leprosy.

FIG. 1.—Portion of a diseased *median nerve* removed at the autopsy of the case marked A. L. 3 (Anæsthetic Leprosy), in the author's first Memoir. × 350 diameters.

a, a. Two nerve-tubules imbedded in the new material and greatly altered by its accumulation; sometimes they are emptied as it were by compression, and their wrinkled sheath alone remains; at others they are more irregularly affected, being dilated and contracted at intervals and have a corrugated aspect.

The new deposit is but scantily provided with nuclei; these vary somewhat in form, are delicately granular, and their long diameter may be $\frac{1}{2000}$th in. or more. The addition of acetic acid does not cause this substance to swell. There is no evidence of elastic-tissue fibres. Here the appearance may be distinguished as "hyalin-fibroid."

FIG. 2.—Portion of *tumefied skin* from the left cheek. Case described as M. L. No. 1 (Mixed Leprosy, anæsthetic and tubercular), in the above Memoir. × 300 diameters.

The new material (here "hyalin-granular") presents a homogeneous aspect, is disposed in layers, as would appear from the position of the nuclei or granular masses; these are of a yellowish colour, granular aspect, and very irregular form; in some cases are indications of a nucleus, but commonly the cellular character is not apparent. In isolated places the material seems to occupy the intervals between bundles of the connective tissue, as if resulting from effusion there. It is not so readily acted upon by acetic acid as is ordinary white fibrous tissue. Æther has little effect.

A portion of tumefied skin from the leg of the same case displayed a similar appearance, but the indications of free cells or nuclei in the new material were more evident, and these cells were sometimes of stellate form or branching into fibres; the granular masses were fewer.

a. Free cells. Diameter $\frac{1}{2000}$th in. to $\frac{1}{1000}$th in. The granular masses are usually of greater length than this.

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Fig 1

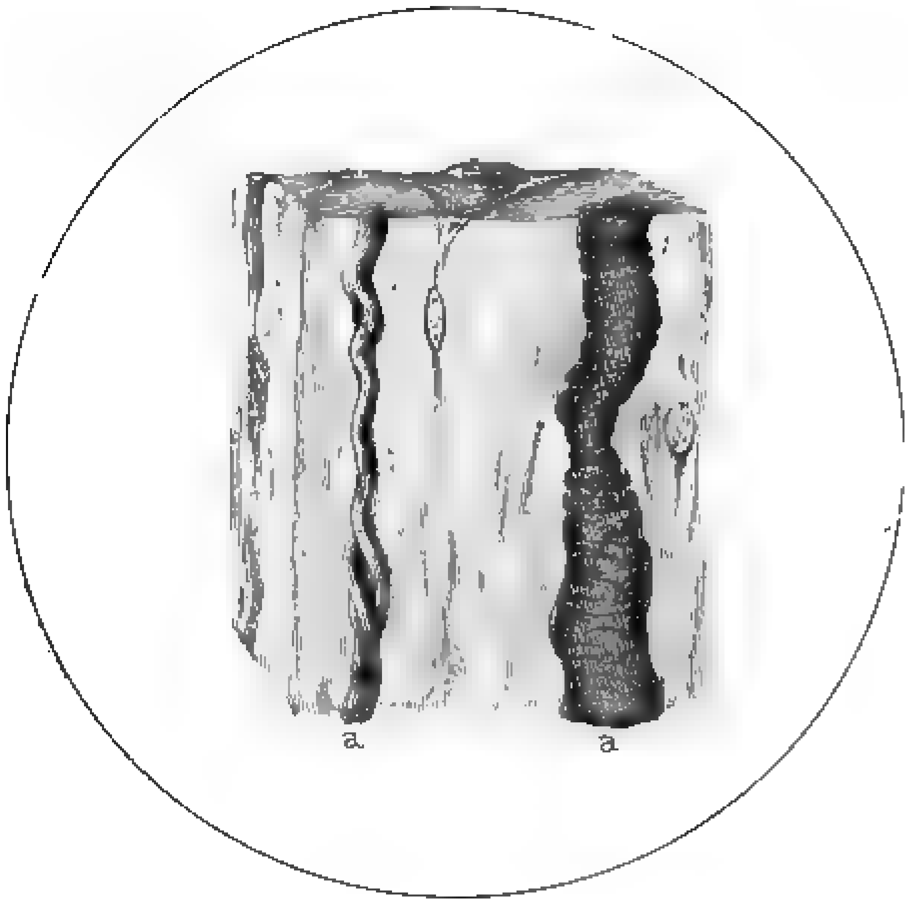
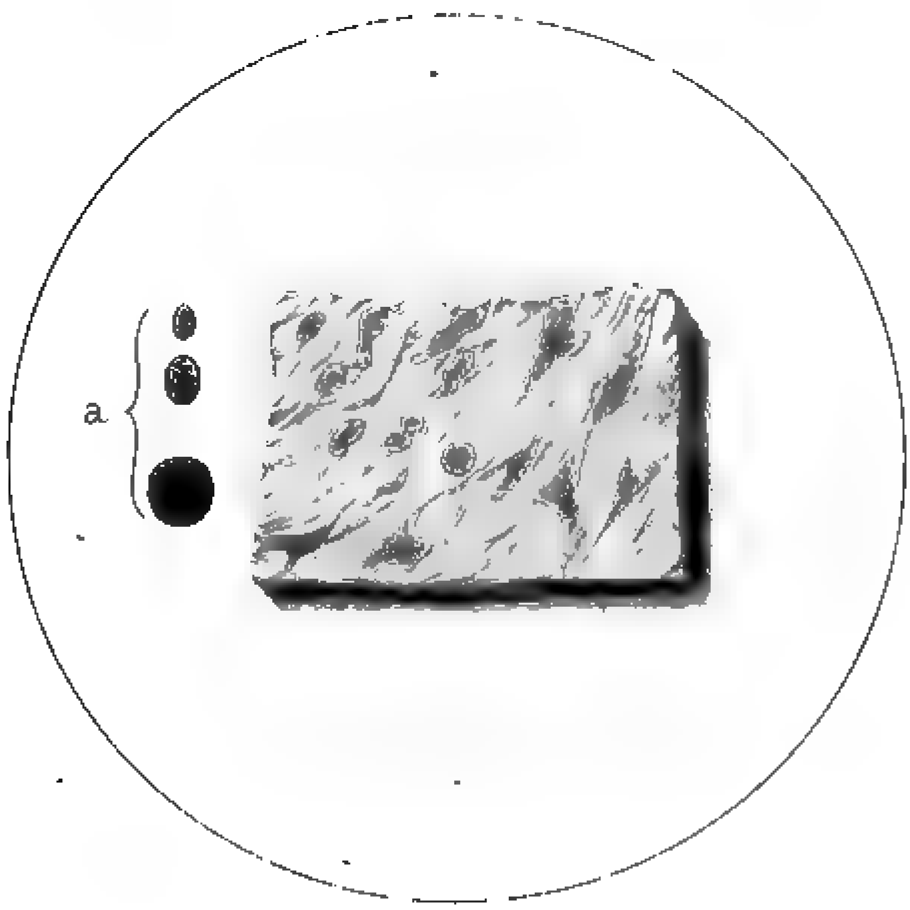


Fig 2



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ON THE

ELEVATED HEALTH RESORTS OF THE SOUTHERN HEMISPHERE.

WITH SPECIAL REFERENCE TO SOUTH AFRICA.

BY

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WORKS on special health resorts, and particularly on those of Europe are almost numberless; but it is not easy to obtain reliable information regarding the effects of long sea voyages, or about the sanitary conditions of our more distant colonies. Having accumulated a good many facts concerning the Australian and South African colonies I venture to hope that some of these may advantageously be laid before the Society. A collateral advantage of the inquiry is that in studying the meteorology of a large continent like that of Australia or South Africa, in which the disturbing causes are less marked than they are in our own little island, much light may occasionally be thrown on the general question of wind, humidity, and the like.

In making a classification of climates the question of latitude is, of course, of prime consideration, and if side by side with this, the elevation of the district be taken into

account, we arrive at certain conclusions which have been laid before the Society by Dr. Hermann Weber in his interesting communication "On the Treatment of Phthisis by Prolonged Residence in Elevated Regions" (see 'Transactions,' vol. lii, p. 225), in which paper he shows that calculations have been made, based on the bills of mortality, for determining the elevation at which immunity from phthisis may be secured in every degree of latitude.

I pointed out in the debate on Dr. Weber's paper that this spot had reference rather to the isothermal lines than to the lines of latitude. Further than this, and without denying the value of the general principle, it appears that to judge of a place as a health resort with regard only or chiefly to its altitude and its latitude involves a fallacy, inasmuch as these form only two out of many elements which go to make up its climate. The range of temperature in summer and in winter is by no means constant at places of the same altitude and latitude, or even at the same altitude and isothermal position. For the range of temperature in summer and in winter varies greatly in places in these respects similarly placed.

Again, the question of dampness and dryness in winter and summer; of rainfall, and its distribution over the year; these and many others are determined less by the latitude and altitude than by the physical geography of the country and of the site which needs to be studied as regards each place.

Happily we are year by year becoming better informed as to the meteorological state of remote places; and the reports published day by day in the United States, showing the direction and strength of the wind, amount of cloud, moisture, barometric and thermometric readings, &c., cannot fail to lead to very valuable results.

The important matter of dryness or humidity is often influenced and controlled by far distant causes. Thus, the far off Alps, and even the deserts of Africa, affect the climate of Italy by causing the Mistral, Tramontana, &c., and also the nearness or distance of ocean tracts, and whether the preva-

lent winds blow to or from the ocean, and the character of the winds consequent on their being land or ocean winds. Again, within and for many degrees outside the tropics the monsoons and trade winds greatly affect the question of dryness and humidity, and these are influenced by the general distribution of land and water over the globe. There are many places which though elevated high above the sea level are so situated as to be damp and airless, and possess many of the characteristics of low-lying swamps.

It seems then that altitude alone, or even altitude and latitude together, can but form a part of the several conditions which go to make a place fit or unfit to be a health resort.

Let us, instead of comparing places lying on the same line of latitude in the northern and southern hemispheres, compare the places lying on the lines of equal temperature —equal temperature, that is, at the sea-level (see map). We observe that the equator of heat crosses Java, Sumatra, Madras, Aden, Sierra Leone, Panama.

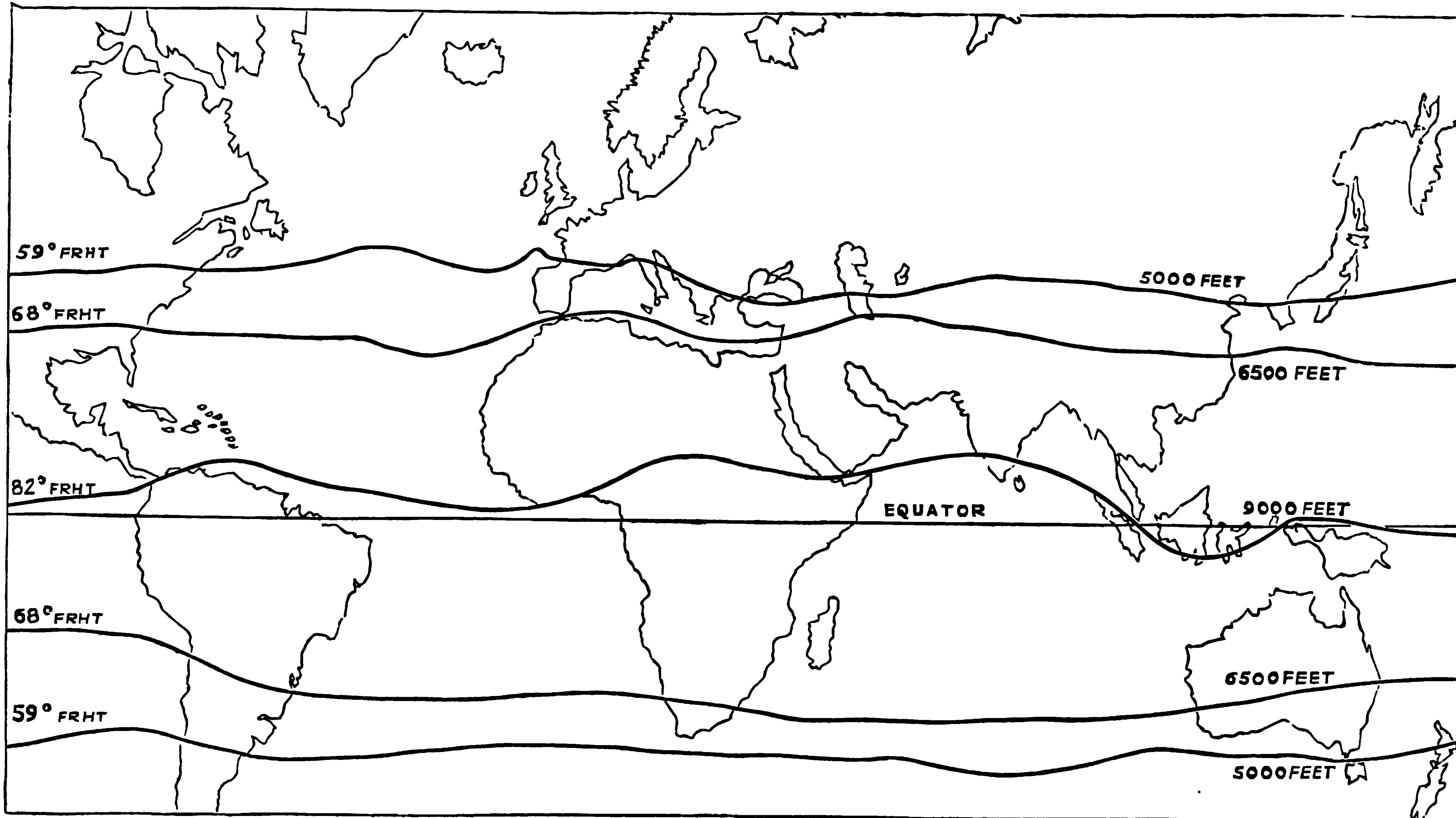
The 68° Ft., isothermal line, N. lat., cuts Beyrout, Malta, Algiers, Madeira, and South Carolina. In the southern hemisphere the corresponding 68° line passes to the northward of Sydney and of Natal.

The line 59° Ft., north, crosses Japan, Pekin, Constantinople, Nice, Pau, Halifax; and 59° south crosses New Zealand, Melbourne, and the Cape.

The assumed line of immunity from phthisis at the equator is drawn at 9000 feet.

For the mean annual temperature 68° Ft. it is drawn at 6500 feet, and for the mean annual temperature 59° Ft. at 5000 feet.

Other lines might have been added, marking different immunity levels, but those shown in the accompanying diagram are sufficient to indicate the general principle involved.


59° FRHT
68° FRHT
82° FRHT
68° FRHT
59° FRHT
5000 FEET
6500 FEET
9000 FEET
EQUATOR
6500 FEET
5000 FEET

It will be seen that these isothermal lines which are drawn on the map so as to pass through all places having the same mean annual temperature, differ considerably from the lines of latitude, diverging more and more from them as they recede from the equator.

The southern hemisphere, having much less land than the northern, especially in its torrid and temperate zones, has a lower mean annual temperature. Hence, it appears probable that in the southern hemisphere a much lower elevation for each parallel of latitude is sufficient for securing immunity from phthisis than in the corresponding parallel of the northern hemisphere.

It is not the object of this communication to deal with those health resorts available for persons suffering from advanced disease who are unable to bear any exertion or exposure. For such, if home is to be deserted, our own shores give us Torquay, Bournemouth, Ventnor, and Hastings; or if the Riviera can be reached, Mentone, Cannes, San Remo, &c., have each their advantages. The problem where to spend the month of May is really a difficulty, for then it is too hot in the Riviera for health or comfort, in England it is too unsettled, and in Switzerland too cold.

Pau owes its remarkable stillness and softness to the elevated plateau which shelters it from north and east. Its rainfall (forty inches) is half as much again as that of London (twenty-seven inches), but the soil is dry and exposed iron does not rust there.

Rome must scarcely be named as a health resort while fever continues so rife there.

The climate of Algiers, intermediate between that of France and the Tropics, benefits some. The air is pure and cloudless, the twilight short, and there are great diurnal variations. The ground slopes upward from the sea, and there are houses built at a height of 400 feet, having gardens which give shade from the glare of the sun.

Compared with Cairo and the Thebaid, Alexandria is moist and undesirable. The whole of Egypt is dry and bracing,

but too hot and dusty. The expense is also great, three months spent there costing as much as a year in a colony.

The exceedingly dry climate of Sinai and Palestine is certainly of value in many relaxed conditions, but the sand and glare are very trying, and a diurnal variation of 40° Ft. occurs.

Beyrout has a delightfully mild, dry climate; care is needed there during the short rainy season of January and February, lasting about three weeks and for ten days in November.

At Malta, Corfu, and in Sicily I have had patients who spoke favorably of each.

Central Spain is high, dry, and bracing, but the sudden changes of temperature there are trying, and in its present state of transitional republicanism, it is almost unsafe.

Of the Swiss resorts it is needless to speak, for they have been ably dealt with by many and are scarcely fitted for prolonged residence. Of the salubrity of Arolla (7000 feet) above Evolena, one of the less known mountain stations, I can speak with confidence, six of my patients having there gained great benefit. The climate of the Lake of Geneva seems to illustrate the statement of Sir James Clark that the neighbourhood of fresh water is bad, humidity being of all qualities of air most injurious to human life.

The distance of a health resort from England is not a matter of great consideration, for facilities of locomotion are so marvellously increased that it is as easy now to go round the world as it was at the beginning of the century to visit Spain or Italy. The public is beginning to realise, too, that in a disease of long standing, or in a condition in which hereditary tendency to disease is strong, little permanent benefit can be expected to result from a few months spent in a health resort, and many are now ready to accept the verdict of the physician that two or more years spent in a well-selected climate can alone be counted upon as likely to secure permanent amendment or established health. The depressing influence of being surrounded as at Cannes or Mentone by respirators and Bath chairs is also avoided in our colonies.

So great is the universal testimony in favour of sea voyages that a proposal has more than once been made to take a party of health seekers for a six months' "sanitary cruise" in a first-class clipper. Cadiz, Gibraltar, Malaga, the Canaries, St. Helena, and the Cape of Good Hope being the points mentioned to touch at in the trip. If the invalids could be kept from serious sickness and in good humour with one another, such a cruise might be a success. The vessel must be comfortable and well found in every way. Many instead of gaining have lost ground in consequence of the bad food and accommodation on board ship.

It has been well observed that in going in a sailing vessel through the trade wind region to the Cape, a cabin should be chosen on the starboard side where the comfort of an open port is more often secured during the day than on the larboard side, which is often under water or within reach of the waves.

The cruelty of sending a delicate invalid with afternoon fever and advancing disease on a long voyage cannot be too strongly insisted on (see Case 11 in the Appendix).

The voyage round the Cape (lat. 35° S.) is generally more favorable than that round the Horn (lat. 56° S.), but the Pacific Steam Navigation vessels now go through the Straits of Magellan and thus avoid the storms and risks of icebergs. The trip from Valparaiso to Panama is described as like a yachting cruise, so smooth is the sea and so bright and sunny the surroundings.

The voyage to Madeira or to the Azores is long enough for discomfort, but not long enough to secure the real benefit of the sea. It is not until the Bay of Biscay is crossed that the landsman begins to enjoy life on board ship.

Cape Town, the first place reached, has many advantages to the health seeker. It is, however, subject to violent "sou'-westers." The town lies low by the water side, is confined and badly drained, and very hot from reflection from the Table Mountain (see Case 1 in the Appendix).

Wynberg, about nine miles off on the line of railway, is

very healthy and pretty, with forests of pine, oak, and silver-wood. It is a most desirable place in summer for an invalid, though in winter the whole region is damp and rainy.

Graham's Town (1800 feet) is pleasant, somewhat cooler than Maritzburg in Natal, and with less constant sunshine in winter; the country round is healthy; the society pleasant, and the shops well provided. A medical friend, now at Port Elizabeth, finds the place healthy, though in the classified list of admissions to his hospital almost every known disease is represented. Graham's Town is to Port Elizabeth what Maritzburg is to D'Urban.

Natal lies between the 28th and 32nd parallels of south latitude. It has 150 miles of sea coast, and is 800 miles distant from the Cape. The mail steamers take thirty-seven days, and sailing vessels from fifty to seventy days, in reaching it. It has been described as a series of steps or plateaux rising successively from the sea to the Drakenberg mountains; but it is rather a succession of ridges and depressions, the ridges being each higher than the last as you go inland.

Proceeding inland from Maritzburg the land rises rapidly another 1800 feet, and then declines gently 300 feet or so to the next river valley, that of the Umgeni, and then again rises rapidly more than 2000 feet, until at seventy miles from the sea it is about one mile high. This elevated upland offers a good illustration of what has been said above. The warm moisture-laden breeze, blowing very commonly from the south-east, impinges on the rapid slope of the highland, looking south-east. The air is suddenly cooled, and at once part of its moisture is condensed and deposited on the rising slopes of the hill as cloud or mist. But, on proceeding a little further inland you leave the mist behind, the air is again clear, and the sky bright. This frequent deposit of mist and cloud upon the seaward mountain slope determines the speciality of its climate, as compared with that which is found a few miles further inland on the same plateau. It is thus that the so-called "

formed upon "Table Mountain," near Cape Town. A white cloud rests upon the top of the mountain, and hangs down over the edge of the precipitous sides, somewhat like a tablecloth, while all around the sky is bright and clear.

The leading peculiarity of the climate is that in winter it has almost constant sunshine and little rain, with abundant rain and cloud in summer, so that the mean temperature of the winter is agreeably raised, and of the summer depressed.

At Maritzburg the mean winter temperature is 60°, and summer 69°, for the whole year 64·7°; that of the coast being 4·5° higher.

The hot land-winds often raise the temperature to 85° or 95°, but these winds are followed by rain, and are less trying than the hot dusty winds of the Australian Colonies, which render exertion in the open air very trying and difficult.

The accompanying table, prepared by Dr. Mann, shows the average rainfall at Natal, which has been placed in parallel columns with that of London, Melbourne, and Rome.

Mean rainfall for a year at—

	London.	Melbourne.	Natal.	Rome.
January	1·56	1·36	3·56	3·62
February	1·45	0·95	4·33	3·38
March	1·36	1·60	3·11	4·52
April	1·55	3·13	1·40	2·95
May	1·67	3·67	1·02	1·89
June	1·98	2·41	0·12	0·11
July	2·44	2·18	0·25	1·18
August	2·37	3·61	1·12	3·58
September	2·97	3·27	1·16	9·37
October	2·46	2·54	3·97	4·13
November	2·58	4·27	4·78	4·08
December	1·65	1·86	5·21	4·01
Total for year	24·04	30·85	30·03	42·82

It will be seen that in Natal the chief rainfall is in summer, *i. e.* the months of November, December, and January, and it is in this respect especially that Natal is superior to the

Cape Colony, in which the summer is dry, and the winter rainy.

Among the Kaffirs, strumous and phthisical conditions are much less common than among the English poor. The medical men of the colony, who have reported on the subject, all speak of the remarkable immunity from these diseases among the English residents, and imported disease becomes latent in a large proportion of cases. The climate is certainly conducive to the longevity of those who come to it in middle life. The mortality among young men is (as in all colonies) somewhat large, but this is attributable to the rough life and unsettled habits of many colonists.

The Rev. Dr. Callaway's Mission Station at Springvale was selected by him as a specially salubrious spot, and in winter it has all the requirements of a health resort. He has a branch establishment at "High Flats," about 1000 feet higher, for the summer's residence of invalids who may be under his care. He has proposed to build a commodious sanatorium, where well-to-do invalids, besides the poor Kaffirs, could be under his skilful treatment. A grant for its maintenance is under the favorable consideration of the local government.

Springvale appears somewhat too near the coast, and its elevation (2500 ft.) is not quite so great as one would desire. Still there can be no doubt of its salubrity during the winter months.

The geological formation * of the Town Hill, behind Maritzburg, consists of sandstone and shale, containing coal, wood, and reptilian remains, but the "Table Mountain Sandstone," which is supposed to be Silurian, is met with on the coast and intervening ridges, overlying granite, gneiss, or porphyry. The town of Maritzburg is built upon shale, broken around by sandstones and dykes of trap. The special "plateau" character of the country is due to the extensive presence of the Table Mountain sandstone, which always occurs in horizontal beds. The Free State and whole

* See coloured section in Griesbach "On the Geology of Natal," 'Quart. Journ. Geolog. Science,' Dec. 1870, vol. xxvii.

interior of South Africa consist of limestone intersected or covered by amygdaloid deposit.

The country about Maritzburg is healthy, and well suited to delicate people; the air is drier than on the coast, and the evenings cooler, but the moist influence of the Indian Ocean is still felt over much.

The country between Maritzburg and the Mooi River Hills is even more remarkably healthy. White people work in the open air, very much as in England, without suffering. This is far from being the case on the coast lands. The alternations of temperature, however, at all seasons, and the sudden changes from wet to dry in summer, as the sea or land winds prevail, are apt to try persons of weak nerve or irritable mucous membrane. An alternation of temperature amounting to 40° Ft. in the twenty-four hours is no uncommon occurrence.

The temperature of the narrow strip of tropical coast land is from 5° to 10° above what might be expected in that latitude, owing to the influence of the Mozambique current —the "African Gulf Stream." Healthy people thrive on this tract, which is free from epidemics, but weakly ones find it wearing and relaxing, remittent fevers and biliary derangements being not unfrequent.

The coast lands at the northern border of Natal are subject to a form of low fever, probably due to bad water. The fever becomes more frequent and more virulent as you go northward into Zulu land. The peculiar entozoon (*Hematobium Capense*) described to this Society by Dr. John Harley specially flourishes there as it does in Mauritius, where the climate is very similar.

The greater part of Natal may be considered as somewhat too relaxing for a perfect climate.

Owing probably to the rapid changes of temperature just referred to, and to the amount of evaporation going on for the greater part of the year, biliary derangements are common, but rheumatic affections are benefited, probably by the free action of the skin. The climate is good for persons with complaints of the throat and chest, those liable in England

to bronchial affections being here surprisingly free from them. (See Cases 5, 6, 7, and 9.)

It appears, on the whole, better suited to *poitrinaires*, to borrow a French word, than to other classes of invalids.

During the winter may be seen the often snowy tops of the distant Drakenbergen. These mountains, which form the broken edge of the whole of the plateau of Southern Africa, rise here and there to a height of 9500 feet, the general ridge is between 5000 and 6000 feet, one peak being 12,000 feet high. Taking a course irregular, but, on the whole, nearly parallel to the sea coast, they shut in the lower lands of Natal and the adjacent territory, as with a gigantic wall, buttressed with its offsets.

Travellers describe the sudden change in descending in summer time from the great plateau beyond the summit of these mountains into the steaming region below. The portion of the plateau immediately beyond the north-west frontier of Natal is called the Free State, or the Orange River Republic, and it is to this region that I would now refer.

Of the moisture which is carried inland from the Indian Ocean by cloud and wind a very large proportion is intercepted by the series of slopes and high ridges which lie between the main chain and the ocean. Of the remainder a still further portion falls upon the slopes of the Drakenbergen. The residue has to supply the wants of a vast space of territory lying beyond the main chain towards the Atlantic Ocean, a distance of many hundred miles. Such results as we might expect from a consideration of these data are found to exist in fact.

If we draw a line from the heights of the Drakenbergen towards the interior of the great continent, we have first the Free State, a dry, pastoral country, with few rivers, and these swollen only at uncertain intervals—devoid of timber and forest, with clear sky and bracing air. Passing northwestward beyond the Free State the climate becomes yet drier and the land more sterile, till we reach at length the

rainless, and utterly arid, barren country of the interior towards Namaqua land.

A theory has been developed by Mr. Shepstone (Secretary for Native Affairs in Natal) to account for the formation of the Central South African table land. This plateau, it is thought, was at one time a vast inland sea, surrounded by the Drakenberg and other mountain chains. Upheaval has occurred at some distant period, and the waters have been disgorged through the Limpopo and the Orange River.

The point, then, to be sought in this long reach of country is that at which neither humidity nor dryness is extreme. We believe this point is to be found somewhere near the capital of the Free State.

The average elevation of the whole Free State is about 5000 feet above the sea level.

Bloemfontein, the capital, was built and laid out by the African-Dutch Boers, and has long straight streets, and abundance of gardens and watercourses. A few years ago there was much difficulty in obtaining suitable provision for an invalid (see Case 9 in Appendix), but the increased and increasing prosperity which has followed in the wake of the diamond discoveries has improved all this, and when the long journey in ox waggons is superseded by the now hoped-for railway, we may expect that this almost inaccessible region will be brought within the reach of a class of invalids to whom it is at present closed.

The climate of the Free State is extolled by all as exhilarating and health restoring. Many go there from the Cape and Natal by the advice of the local doctors, and, as a rule, return to their less bracing homes greatly invigorated.

The diurnal variation of temperature is not so great in the Free State as in the Engadine. Snow and frost are very exceptional. It has been maintained by Dr. Burney Yeo that a short stay in an elevated resort is better than a prolonged residence there. This, though probably true as regards the Swiss mountains, is not so in the South African table lands at the same elevation.

The country slopes gradually from Bloemfontein, which

seems to be the most eligible part of all South Africa for those suffering from chest complaints and general debility, to the junction of the Orange and Vaal Rivers, and the tract occupied by the diamond diggers. Grass gives place to prickly shrubs, clay to sand and chalky lime. The climate here is excessively dry, and in summer overpoweringly hot: by day ranging to 100° F. in the shade. The nights, however, are on account of the elevation comparatively cool, and there is generally a lively breeze.

For all who are not obliged to live in a canvas town of 40,000 inhabitants, reeking with sewage and other garbage, and nourished on beefsteak and rum, the climate is very salubrious, especially in the winter, and many derive benefit from six months' sojourn under canvas; there is, however, some risk of fever, due to the tainted air and water of the camp rather than to the malaria of the climate.

India, with its wonderful mountain ranges, affords many elevated health resorts, which are now accessible even to English invalids.*

The finest ships in the world are now running from England by Suez to India in from twenty-five to thirty-five days, and there are railways to the foot of the hills. There cannot be a greater contrast than between the climate of the hill stations of India and that of England. The climate is in the highest degree stimulating, like that of Australia, and many invalids who have friends in India may naturally incline thither rather than to the Australian and South African colonies. The attendance of a good Indian servant may partly make up even for the absence of friends, as the devotion of a native "boy" to his master amounts almost to idolatry, whereas in a colony the difficulty of obtaining a tender and devoted servant is often very great.

It is only in a meteorological sense that any part of India can be regarded as belonging to the southern hemisphere. It will be seen on referring to the map that the line of equatorial heat passes north of Ceylon and of South India.

* See the recent articles by Dr. Druitt in the 'Med. Times and Gazette,' June, 1873, pp. 580 and 634.

The chain of the Himalayas is to the northward of this line, and only a passing allusion must be made to Dalhousie and to Darjeeling, the elevated health resorts of the Calcutta invalids, where the peculiarity of the climate is its remarkable equability of temperature. A friend, who kept a registering thermometer in his verandah, tells me that for four months he never registered a higher temperature than 64° or a lower than 62°; summer and winter being equally unknown. There is, however, much fog and drizzling rain. The average rainfall is 140 inches.

Chillong is the newest popular health resort in this part of India, but is very inaccessible.

The difference in the rainfall of the various hill resorts of India is striking. In one, Cherapongy (lat. 24° N.), the rainfall is from 600 to 800 inches, whilst at Chenar in the Punjaub (lat. 34° N.) it is only four inches.

The Neilgherries, lying south of the equatorial heat line, rise abruptly from the plains to a height of from 6000 to 7000 feet, the highest point being 8760 feet.

The foot of the hill may be reached from Madras in fifteen hours by railway, and in half an hour after leaving the station, whether you travel in bullock waggon or palanquin, you escape from the heat of the plains. From Bombay coasting steamers go to Beypoor, whence the railway takes passengers to the foot of the hills.

The Suez route to India cannot be recommended for very sensitive invalids (see Case 2), and the voyage round the Cape is as long as that to Australia; indeed, the Australian coast is often sighted before turning northwards to Calcutta or Singapore.

Ootacamund, the principal station of the Neilgherries, is at the centre of the table land (7400 feet elevation) and quite European in climate and scenery. Heavy rains occur during the monsoons (July and August), 400 inches being a frequent annual rainfall. At Conoor, a few miles off (elevation 6400 feet), there is but little rain, cloud, or mist. In this latitude fever range is calculated to be at 4500 feet elevation. The whole of the Neilgherry range is therefore much above this. The

base of the hills is surrounded by dense jungle from six to twenty miles wide, and extending 2000 or 3000 feet up the side of the hills. There is then an open space a mile or two wide, and above this again woods like those of temperate climates. There are swamps at the summit, but no miasma. In the hottest part of the year clouds prevail, and there being no snowfields, as in the Himalaya range, this climate is very equable.

When the Neilgherries were first resorted to many English lost their lives, rapidly fatal inflammation being set up by exposure, the contrast to the low lands being too great, but with caution this risk may be avoided. The healthy appearance of the residents is a striking evidence of the salubrity of the climate.

Newera Ellia, the sanatorium of Ceylon (5000 feet), eighty miles from Kandy, the capital, is much resorted to from Calcutta and Bombay, especially by ladies with pulmonary complaints. It is, however, difficult of access, and the cost of living in proportion to the comfort obtained is great.

Immediately above Penang there is an elevated sanatorium which may be reached in about two hours from the port.

As regards the climate of Australia great diversity of opinion has prevailed, and an angry controversy has been raging in Melbourne on the subject. The climate, speaking generally, is highly stimulating owing to the prevalence of ocean breezes. There are no lofty mountain ranges, and really elevated health resorts are wanting.

Western Australia is superior, as regards general salubrity, to the other Australian colonies, and efforts have been made to establish a sanatorium there, but a prejudice exists against it owing to the fact that it was made (in 1851) a penal settlement. In early chest disease the effect of the climate has proved in three instances that have been brought under my notice strikingly beneficial, but advanced cases are rapidly hastened to a termination. Heart disease is reported to be very common.

Adelaide in South Australia is dry and dusty. Its rainfall is twenty-one inches, but Mount Lofty (2000 feet) behind the

town is dotted with suburban villas which are both pleasant and healthy.

Melbourne itself is hot and dusty. The large mortality from consumption in the town is attributed by Dr. Thomson, in his work on the Victoria climate, to the fact that much of the town is built upon swamps.

Some of the up-country stations are cooler than the town and very healthy. Parts of Gipps' Land are elevated and salubrious.

Sydney is bright and pleasant in winter, but decidedly too hot in summer. The climate is dry, though the rainfall is fifty inches. The soil is sandy, and the hot sirocco-like winds are very trying. The hurricanes from the south, which are called "southerly busters," and from the dust they bring, "brick fielders," are very trying to sufferers from chest disease.

The mean annual temperature is 65° (London 58°), that of the summer being 74° (London 60°), and of the winter 55° (London 37°).

Goulburn and Bathurst stand high (nearly 2000 feet) and are bracing. People come thence into Sydney with ruddy cheeks. The Murray and Murrumbidge regions supply a fine race of squatters. Betchworth, the capital of the Murray river district, is 1750 feet above the sea level.

A two hours railway journey from Melbourne brings the traveller to Kyneton or Woodend (height 2000 to 3000 feet). Ballarat is 1500 feet, and much sheltered. The Darling Downs (2000 to 3000 feet) afford a splendid summer climate. Judging from the sensations of travellers it would seem that an elevation of 3000 or 4000 feet affords as invigorating an influence as is to be gained at twice the altitude in the northern hemisphere at a corresponding parallel of latitude.

Queensland, though somewhat hotter, is much like Natal in climate. The wet season coming in summer produces the steaming tropical heat which gives rise to rank and luxuriant vegetation, and a form of colonial fever, ague and dysentery combined, not unlike the fever of Zulu land, is often met

with. Still, as sheep thrive there it is evident that there are tracts in the colony of Queensland where other characteristics prevail.

On comparing the climate of Victoria with that of Natal, while the mean temperature appears to be much the same, a very important difference exists between the two climates, viz. that in Natal the wet season occurs in summer, in Victoria in winter. The result, so far as the lower animals are concerned, is, that the Australian climate is more favorable to health than that of Natal, and the same is possibly true as regards human life. This conclusion is not, however, so evident as might appear at first sight, for an effect of the rainless winter is, that in Natal the grass is most scant in cold weather when it is most needed.

The Natal winter is, on the whole, more healthy for invalids than summer or winter in Victoria, and the Victoria summer probably more healthy than the summer in Natal.

The climate of Victoria taken altogether is drier (and probably hotter) than that of Natal. Vegetation shows this. The blue gum tree of Australia increases in height and bulk more rapidly than in its own country.*

In Australia, South America, and in the Swiss mountain resorts deaths from acute diseases or catarrhal fever, pneumonia and pleurisy, are very common, while those from chronic diseases, and especially consumption, are rare. The contrast between the prevailing maladies of England and Australia, being similar to that pointed out by Dr. King Chambers as existing between the English and Italian discases,† we find that diseases dependent on degeneration of tissue are as rare in Australia as in Italy.

In the Northern States of America phthisis is very fatal and frequent, whereas in the Southern States it is rare, while pneumonia and bronchitis are very common.

Tasmania or Van Dieman's Land has a very delightful sunny climate. The weather is variable, though less so than

* See Dr. Callaway's 'Report of Spring Vale.'

† *Vide* his 'Climate of Italy,' p. 47.

in England, there being no sudden extremes of temperature. English comforts are cheap and abundant.

At Hobart Town the sea breeze is sometimes strong, though it does not last long (see Case 12), and may be escaped on going inland.

The Tasmanian climate has a great and well-deserved reputation as a health resort for broken-down Anglo-Indians.

It is a very exciting climate, and many sudden deaths occur to persons in the prime of life.

Dr. Bird, in his work on the 'Australian Climate,' speaks of Launceston as the Pau of the southern hemisphere. Its situation is, however, low and swampy.

There are no *really* elevated towns or villages in the island. The Tasmanian mountains do not rise higher than 3000 or 4000 feet.

New Zealand, on the other hand, is very mountainous, some of its peaks rising to 14,000 feet. These are snow clad, and have extensive glaciers. Being like the Sierra of Spain there are no elevated table lands or settled accommodation for invalids in the uplands, *i. e.* above 3000 feet. The islands extend from latitude 34° to 46° S., and the varieties of climate are naturally great. South-east winds prevail, and the climate is generally bracing.

Wellington, lying on a point between the two islands, is perhaps the most windy place in the world (see Case 7).

The climate of Cook's Straits is very salubrious, and Nelson lying in its sheltered bay, has a charming climate. The unvarying character of the climate makes occasional change desirable.

Auckland, the capital of the Northern Island, a cheap and prosperous district, is moist, warm, and relaxing, but with boisterous winds. Although strong, the winds are usually 2° or 3° warmer than the atmosphere generally, and, owing to the absence of neighbouring deserts, there is nothing like an east wind to be met with.

A medical friend, who for a quarter of a century has been settled in Canterbury district, describes the climate as tem-

perate and delightful. He has all the consulting practice of the neighbourhood, but the place is so healthy that except in the treatment of accidents (he has himself broken seven bones and his daughter two) he has little or nothing to do.

Slight earthquakes sometimes occur. Speaking generally, Otago and Auckland have too much rain, and Canterbury and Wellington too much wind.

The elevated South American health resorts are too numerous to mention. Each important town of the Pacific shore has its elevated sanatorium.

Though some (as Arequipa) are very cold, others are spoken of with great praise, and doubtless have many advantages over the hot and crowded ports below. The country is highly volcanic, and exposed to frequent earthquakes, and in many parts the government is neither stable nor satisfactory. There are few English doctors and, indeed, few persons speaking English there. Rain seldom if ever falls, and there is no dew. Cultivation is entirely dependent on irrigation, the water arising from the melting snow on the Andes. As seen from the sea, much of the coast is thoroughly barren and without any sign of vegetation.

Piura above the port of Payta in Peru (lat. 5° 9′ S.), has a wonderful reputation especially in cases of chronic syphilis.

At Lima, on the equator, removal to the mountain health resorts (9000 to 12,000 feet above the sea level), is regarded as preventive and curative of hæmoptysis and phthisis (Archibald Smith). In the dry climate of the West Coast of South America, the deaths due to drink are very numerous; the water drinkers dying of dysentery and the spirit drinkers of delirium tremens.

Having had full information from a number of different sources, it has been possible in the foregoing paper to compare observations made, and opinions formed in the various parts of South Africa and Australia; whereas the information obtained from the other countries, and especially with regard to the South American elevated health resorts, has been of a less complete and detailed character.

Cases of recovery from lung disease in the various stages and varieties are happily to be met with under all circumstances and at every place. Among hospital out-patients subjected to conditions little favorable to such a result, it is easy to select many instances of regained health even from advanced disease, and among the in-patients at a hospital such likewise occur, so that the description of instances of phthisical disease undergoing arrest and ultimate recovery under altered climatic conditions could only prove that what is true of hospital out-patients badly housed and fed is true also of those who have travelled the world over in search of health.

It seems useless, therefore, to append full details of one or two cases, nor have the number of cases under observation been sufficient for a statistical table giving the percentage of recoveries to attacks in different countries.

The impression that a larger proportion of those who go to the colonies with chest disease do well than of those who remain, is strong in my mind as in that of most authorities on the subject. Yet this opinion may be strengthened by the fact that the cases that go out at our bidding are carefully selected, that those who go out against our will and notwithstanding our warnings rarely write to confess their error, nor are we always informed when death occurs.

The following cases, though too meagre to be of much scientific value, may not, it is hoped, be altogether devoid of interest, as being the grounds on which the general results of this paper have been built.

APPENDIX.

CASE 1.—*Chronic phthisis; life at the Cape useless; voyage out and home injurious.*

A pianoforte toner, æt. 35, first seen September, 1869. Complained during the last twelve months of occasional cough with frothy phlegm and streaks of blood. A tall, fair, anæmic-looking man; tongue white and dry: fauc[illegible]nular and congested; chest sounds normal; weight 8

April, 1871.—Cough frequent and sputa scanty; digestion disturbed; weight 8 st. 4 lbs.

On examining the chest, breathing on the right side feeble throughout, with conducted heart sounds and crepitation at the apex. Left side, breathing blowing (compensatory).

May 4th.—Symptoms relieved under cod-liver oil, good diet and counter-irritation. Respiration still feeble on right side with crepitation at the apex. Left England in August in a sailing vessel (500 tons), caught cold in the Channel and never lost it. Landed at the Cape worse than when he started. Soon after landing had diarrhœa, the town being badly drained, and quickly lost ground. He then went to Wynberg; this proved wet and did not suit him (he now regrets that he did not take a little house there in a garden), so was persuaded to go up to Beaufort,* as being the driest place near. He bought a cart and mule and drove up there in seven days. Beaufort (3000 feet elevation) proved to be very dusty; it is situate in the bed of a river. Although windy it is less so than Cape Town. He remained there ten months; food was insufficient, but he gained flesh. The place was so dreary and monotonous that the doctor advised his return; he got into a waggon and drove down in three days and three nights to the Cape. There he remained a month and gained somewhat though he had diarrhœa and liver disorder. The voyage home in the "Celt" (2000 tons), Union Company Steamship, proved rough and food bad.

On arrival he was much emaciated. Pulse 120; sputa copious, hawked up with difficulty and with some laryngeal discomfort.

Physical signs.—Right side dull, flat; conducted heart sounds and impulse; crackling crepitation to base. Left side, blowing upper lobe; fine crepitation base.

Remarks.—In this case the disease was not advanced or extensive on leaving England, but it developed uninterruptedly in the colony. The patient lacked the energy and self-reliance necessary for "roughing it" in a new country.

* Some details about Beaufort, &c., are given in the 'Lancet,' "The Sanitarium of South Africa," pp. 63-4, July 12, 1873.

Case 2.—*Phthisis; second stage; extensive disease; arrest in Natal.*

A publican, æt. 25, first seen in September, 1867. Cough two years, following severe cold, caught from getting feet wet after a ball; hæmoptysis a month ago; sputa copious and nummular.

Physical signs.—Right side normal. Left flattening, dulness, *bruit de pôt fêle;* gurgling at apex; no natural vesicular murmur; rhonchus and crepitation throughout the lung extending nearly to the base. Pulse 100; tongue white; weight 9 st.; height 5 ft. 8 in. Improved under beefsteaks three times a day, cod-liver oil, nitric acid, and Lint. Tiglii.

March, 1868.—Weight 9 st. 7 lbs.

Physical signs.—Left side dulness; humid crepitation; bronchial breathing; bronchophony; crepitation to base. Cod-liver oil continued, ordered horse exercise.

On April 8th he went to Natal, which he reached in a sailing vessel (500 tons) in eighty-two days; enjoyed the voyage; got daily better; coughed less; gained 7 lb. in weight (10 st. 7 lbs.); arrived June 31st. Spent two months at Durban (Royal Hotel, very good); but cough continued.

On his return, October 26th, pulse 84.

Physical signs.—Right side, respiration blowing; left, dull upper third; dry crackling râles mixed with vesicular respiration; slight sibilus with vesicular murmur in suprascapular fossa; slight, coarse, crepitant râles nearly to base behind.

Dec. 2nd.—Troublesome cough and catarrh. Physical signs unchanged. This patient writes that he is now free from cough.

Case 3.—*Early phthisis; sea voyage; Cape Town and Natal; cure.*

A banker's clerk of delicate family; sister phthisical; at age of twenty-two began to lose flesh and colour, and in the winter had cough with sago-like expectoration containing

coils of lung tissue, occasional streaky hæmoptysis; red streaks on margins of gums; slight dulness on percussion at left apex, with wavy inspiration. A sea voyage being recommended he went to the Cape in one of H.M.'s frigates, improved on the voyage and at the Cape; remained three months at Natal, where he gained flesh and strength and was able to bear exposure to all weathers. The voyage home established the benefit gained.

On examining the chest, wavy breathing beneath the left clavicle is the only noticeable sign of lung affection.

Returned to the bank, in which he is now partner. Has married and has three rather delicate children; he looks thin and pale, but never complains of serious illness, and has not consulted me nor, I believe, any one else, for years.

CASE 4.—*Consolidation and softening of lung; sea voyage round the Cape and sojourn at Natal useless; death.*

A clerk, æt. 25, in wholesale tea trade; small and of delicate build. Father asthmatical. First consulted me in September, 1871.

History.—Cough sixteen months; increased when in the Isle of Man, where he had hæmoptysis to the extent of half a pint; streaky hæmoptysis since; night sweats; has recently lost flesh; tongue pale; complains of sickness and flatulence; no afternoon fever.

Physical signs.—Right side blowing; left, slightly dull at base and apex, with conducted cardiac sounds; crackle on cough and on deep inspiration beneath clavicle.

Sea voyage to Sydney or Natal proposed.

In October there was some bronchial breathing besides the crackle in the subclavicular fossa, with flattening and diminished movement.

Nov. 9th.—Left England for Natal in a sailing vessel, the "Umzinto" (400 tons), pleasant people on board but food coarse; was nine weeks going out, caught cold, and was much depressed. Landed at Durban, found it very hot;

ordered up to Richmond (sixty miles); found it frosty; was never hopeful; got steadily worse and died suddenly from diarrhœa in July, 1872, six months after his arrival.

Remarks.—In this case disease seems to have advanced during the voyage, and was, perhaps, making insidious progress before he left England, but there was no elevation of temperature and it was hoped the open-air life on board ship would have done much; the food, however, was not good and no rally was made from first to last.

CASE 5.—*Chronic bronchitis and emphysema; freedom from acute bronchial attacks; but deterioration of general health in Natal.*

Architect, æt. 56, when exposed to the glare of Egypt, thirty-two years ago, suffered from conjunctivitis, which has proved a trial through life. Went to Australia in 1849, lived in Victoria four years, enjoyed good health on the whole, but suffered from glare and dust. Returned to England in 1854, found climate trying, and determined to emigrate to Natal, where he lived sixteen years, during which time he narrowly escaped sunstroke; suffered from eczema capitis, and deteriorated physically. Had dyspnœa on exertion, but rarely cough or expectoration. Came to England in 1858, when he was very subject to catarrh and recurring bronchitis on slight exposure. Much emphysema; chest dilated, and moving *en masse.* Rhonchus, sibilus, and prolonged respiratory murmur all over.

Examined by Dr. Andrew Clark, who diagnosed chronic fibrous involvement of lung. Being unable to bear the English winter returned to Natal, where he again became free from bronchial attacks, but the dyspnœa never left him, and the somewhat enervating effect of the subtropical climate of Richmond (3000 feet elevation) seemed further to deteriorate tissue.

In England there is much rheumatic sensitiveness, less in the colony, though in the summer rains, when the air is full of moisture, he suffers somewhat. Now in England again, the

emphysema with perpetually recurring bronchial attacks and constant wheezing continues. The blood is so imperfectly aerated that much effort, physical or mental, leads to headache and oppression. The extremities are cold, and there is great sensitiveness of skin to draughts, cold air or cold sheets, also to damp. The digestion is feeble, owing probably to poverty of blood, and power of locomotion small, especially in cold weather.

When bronchial congestion exists, paroxysmal asthma or asthmatic and useless cough is frequent.

Italy with its bright sunny climate suits well for some months of the year, and the waters of Ems had a favorable influence on the chronic digestive disorder which is little amenable to drugs.

In this case the Natal climate certainly holds in check the bronchitic tendency, and allows of much general out-door exercise, but it is possible that it has exercised an unfavorable influence on the tissues, such as is usual in tropical or subtropical countries.

Remarks.—The voyage to and from the Cape had a decidedly favorable effect, bronchitis ceased, and difficulty of breathing lessened, though the latter returned on re-exposure to land influences. This good effect of a sea voyage is remarked by most consumptive patients as real and certain.

Case 6.—*Phthisis, first stage; arrest in Natal coast lands.*

Rev. G. H—, æt. 30, always delicate, obliged to leave his curacy in north of England, and after two or three attempts to return to clerical duty in England went to Natal suffering from established lung disease. (I did not examine him at this time.) Much benefited by the voyage. Settled at Durban, the port, where he has ever since been able to accomplish hard work though the climate is often very hot. When in England last year had not lost his pale, thin, delicate aspect, but all trace of active lung mischief had gone. In England he was obliged to be very cautious of

exposure to night air, but in the colony took no special precautions.

He has married and returned to active life in the colony. Somewhat improved in general tone by a year spent in a temperate climate, though the lung sensitiveness seemed to be again developed when in London.

The last reports of the patient are unfavorable.

Case 7.—*Bronchitis and spasmodic asthma; bronchial attacks absent in coast lands of Natal.*

A missionary clergyman with asthma and general bronchitis, able in the coast lands, the hotter part of the Natal colony, to work for thirteen years at clerical duty, but when in England he suffered so frightfully from recurring bronchitis and perpetual paroxysmal asthma that it seemed doubtful whether he could safely undertake the voyage out.

But since his return he has been able to prosecute his duties, although when in England his appearance—rounded shoulders, gasping breath, with loud rhonchus, sibilus, and prolonged expiration all over the chest, with heart pulsating forcibly in the scrobiculus cordis—made the idea of work impossible.

Another missionary clergyman, an M.D. once well known at Bartholomew's Hospital, was obliged to leave practice in London on health grounds (early tubercular phthisis). From the time he landed at Natal he has been able to work hard and bear much exposure without any return of hæmoptysis or chest symptoms.

Case 8.—*Phthisis, second stage, with hæmoptysis, arrested in Natal and recurring in England.*

A banker; phthisical parents; had cough, hæm[illegible] much loss of flesh, and after two or three trials was [illegible] to give up work at the bank. Went to Natal, but w[illegible] critically ill that it was doubted wheth[illegible] could safely attempted. Rallied much at N[illegible] rapid, and in eight months he came[illegible]

opinion, but on returning to the office the old symptoms returned. Went a second time to Natal with like results, accepted post of bank manager there, and after five years of health, came to England on bank business, but in a week was seized with severe hæmoptysis.

I then saw him for the first time, and found evidence of old disease in a flattened chest on one side, and on the other the physical signs showed the presence of recently effused blood. The progress made in England was slow; as soon as possible he was again on board ship; before reaching Durban he felt nearly well, and now reports favorably, though he has made up his mind that he can never safely return to England.

CASE 9.—*Chronic phthisis, arrest in Natal, return in England; second arrest in Free State; death.*

This case, which illustrates many points alluded to in the foregoing paper, is more fully detailed than the others.

An architect, æt. 21, always weakly; of delicate family. Exerted himself much in stopping a runaway horse, and immediately spat up about a pint of florid blood. Hæmoptysis twice recurred afterwards. Cough commenced from this time, with muco-purulent, occasionally streaky phlegm; night sweats and loss of flesh. Spent the winter of 1862–63 in Ventnor with slight benefit. I first examined his chest in September, 1864. Expansion and resonance good; voice loud on right side; respiration wavy and tubular left. Went to Natal and improved, but did not lose cough; got "jungle fever" when living in a tent during the wet season, and afterwards got tænia and a small flat worm.

When he returned to England the breathing at the left apex was rough and sibilant, and some dulness was detected at the left base, with sibilant and coarse crepitant sounds. Tapeworm removed (head and 20 ft.) after starving twenty-four hours, and then taking ʒij of oil of male fern.

In London, cough so violent and spasmodic that Hastings was tried, and as winter approached, Cannes. The journey to the Riviera tired him much, and the vicinity of the Medi-

terranean prevented sleep; but on retiring to the hills behind the town sleep became possible. In December reported, "Cough and sputa less; appetite good; digestion 'first rate.'"

May, 1867.—Caught cold on the Rhine, with increased bronchial mischief; and such was the craving for Natal sunshine (almost all who have lived in Natal feel that) that the patient determined to return to it. The voyage out lasted seven weeks, and did good; so did Maritzburg; but being advised to take the journey to the Free State, he bought a waggon and started thither.

The journey occupied twenty-four days, and the patient evidently had not strength to undertake it safely; sometimes he could get no food for twelve hours, and once had to go for twenty-four hours with nothing but a rusk and a cup of tea. He described his experiences thus:—"Country very barren; grass withered or burnt up; not a vestige of a tree. Heat in the middle of the day intense; cold at night intenser; yet I never took cold, and if I could have eaten more should have got well."

When at Bloemfontein he wrote—"The air is purity itself; when recovered from the fatigue of the journey I shall pick up fast; my strength is increasing daily. I never cough, except a single fit in the morning. This is a land of extremes, dry weather comes so that your nails crack; the air is hot, close, and motionless, or there is such a wind as to carry you off your feet; the rain comes down in torrents, flooding the rivers and drowning the mail cart and horses."

February 2nd, 1869.—"A change is rapidly coming over my life; health is again improving, and to a more considerable extent than for three years past." A week after this note the patient had an acute inflammatory attack, which lasted till May, when the appetite returned and the cough ceased.

June 22nd.—He notes, "Weather wintry, am glad of warm clothing, and sit over the wood fire. For two days the wind has been blowing a perfect hurricane; the dust affects my chest much; these winds are due in August and September."

July 7th.—"Now the weather is indescribably magnificent; mornings and evenings cool, but the days are glorious, warm, sunny, with an atmosphere unsurpassably breathable, and yet I cough on till sick daily."

September.—"Weather really hot; the warmth has done wonders for me."

October 18th.—"Temperature 90° F. in the morning. Health improving."

January, 1870.—"If a dry climate can ever cause my lungs to heal or arrest the disease, here I shall be successful; but will the inconveniences not counterbalance all these good effects?"

February 8th.—"We have had a ten-days' deluge after a terrible drought; when the rain comes the whole country becomes green in three days. If at a farmhouse I might get better, but in town one can get something to eat; whereas at a farm you get bad bread and tough mutton, and nothing else."

The last note, made a few days before his death, is this—"Day follows day without a shade of improvement, and the heat is most fatiguing."

Remarks.—As the disease in the lungs was in this patient both advanced and extensive, recovery could not be looked for. Nineteen months were spent in Bloemfontein in comparative ease from cough. The great trial was separation from friends and a felt inability to live among them. The patient was exceedingly sensitive to climatic changes, and certainly was unfit for the vicissitudes of a month's waggon journey, but that he survived this and lived so many months is a striking evidence of the salubrity of the climate, and shows that the open-air life, with a fur-lined bed to retire to from sun and wind, is less trying than might be supposed.

From this patient I received many details of analogous cases to his own. Two young men left England in the same ship, both consumptive, and seemingly about equally ill. One remained near the coast, and at first made progress, but soon relapsed and died in fifteen months. The other gradually rallied at Bloemfontein, was eventually able to dispense

with furs and wraps, became a hardy settler, returned to England, married an heiress, and went back to live in the region that brought him health.

The arrangement of a bullock waggon is laborious, and the cost considerable. Kaffir "boys" are often very troublesome, cattle get lost, the waggon, perhaps, sticks in a river or drift, and there is much risk of exposure. With a well-organised team, however, the waggon goes smoothly on its way, and the invalid may lie in bed for days with a pleasant view of the country from his rocking couch, with books, maps, and comforts around him.

Case 10.—*Threatened phthisis; life in Natal and Free State; well.*

A. B—, æt. 19, a tall youth, with soft skin, easily perspiring, and easily tired; lost three brothers and two sisters from lung disease. Consulted me in September, 1870, to ascertain whether he had any tendency to consumption. He had a soft pulse of 84, was prone to catarrh from exposure to night air, and slow to throw off colds. I could detect no decided chest mischief, but advised a voyage and colonial life.

Went to Natal, and subsequently to the Free State to an ostrich farm, but got moped in the absence of all society. Very happy at Maritzburg, and on his return home in autumn of 1872, a sturdy, vigorous, powerfully built colonist, determined to go again to Natal to settle as a silk farmer on the coast. It is, however, very doubtful whether the subtropical climate of the coast will prove as salubrious as the high, dry, and cooler region of the Free State.

Case 11.—*Phthisis, second stage, affecting both lungs; Ceylon useless; death at Aden.*

C. D—, æt. 30, Kentish farmer; phthisical family, first seen in October, 1871; always delicate; married, three small children; much recent anxiety. Ten years ago had per-

sistent cough and loss of flesh, and went to Australia with much benefit. Pulse 100; tongue large and flabby; drowsy after meals; flatulence; debility on exertion.

Physical signs.—Dullish on percussion; bronchial breathing; creaky crepitation in the supra-clavicular and supra-spinous fossæ. Height 5 ft. 10 in.; weight 11 st. 8 lbs.

Went to Jersey; rough passage; found climate very humid; returned no better on November 7th. Cough; expectoration streaky; hæmoptysis. Suggested Egypt for winter, but preferred to remain in his dry sheltered home on "Kentish Rag."

In January went to Ventnor, where he had bronchitis and hæmoptysis; lost flesh and appetite; returned home in May improved. Pulse 94.

Physical signs.—Right side dull, resistent; bronchial breathing; supra-clavicular and supra-spinous fossæ; rhonchus upper fourth back and front. Sick of Ventnor, where all patients did badly in April, and going to Ceylon next winter.

July, 1872.—Cough; expectoration; hæmoptysis; but can ride about his farm chasing bullocks, and walk ten miles at a stretch.

September.—Pulse 100; sputa copious; losing flesh. Right side very dull on percussion; feeble respiration; crepitation almost humid. Left side, sibilus and prolonged expiration, with crepitation at the extreme apex, back and front. Takes cod-liver oil and iron, and keeps skin sore with iodine paint.

Went to Ceylon by overland route. Had bad food on voyage from Suez. Found Colombo very hot in the day, cold and damp in the evening. Did not reach the Ceylon sanatorium at Neura Ellia (5000 ft.), but after a month's trial of the climate determined to return home. Dr. Rowe, of Colombo, advised Mentone, and said that Ceylon was a very good place for threatened consumption, but when the disease was established the very worst. On returning through the Red Sea he was prostrated by the heat, and died on February 18th, 1873.

Remarks.—As both lungs were implicated, and the disease was evidently advancing, the result in this case would not, it was feared, prove satisfactory. But the active life the patient was able to lead in England made the case seem more hopeful. The sad termination of the experiment showed that the trip ought not to have been sanctioned.

Case 12.—*Fistula and threatened phthisis; voyage to Calcutta; life in hills; cure.*

Clerk to a tea broker, æt. 19; mother asthmatic; father cardiac disorder; left school at seventeen, and grew very rapidly. After uncontrolled constipation suffered from piles, followed by fistula, necessitating operation; the incision was deep, as the sinus opened into the bowel, nearly four inches within the anus; healing was very tedious. He lost flesh; perspired at night; had afternoon hectic, and very rapid pulse; and it was feared that lung mischief would be established, and notwithstanding change to the seaside, and vigorous feeding and stimulation, he remained for a year with the unhealed wound.

A sea voyage was advised, and he started for Calcutta, sailing round the Cape. He improved immensely on the voyage, went up to the hills with further benefit, and the return voyage seems permanently to have re-established his health. Having only just reached England, it is not possible to say more, but he is now broad-shouldered, firm, healthy looking; skin whole, and so far as physical signs go perfectly sound. He has returned to work with vigour.

Case 13.—*Phthisis, second stage; voyage to Melbourne of benefit; death on return to England.*

Schoolmaster, æt. 38, first seen in consultation with Dr. M. Mackenzie in January, 1868. Had then suffered from cough two years, following pleurisy; slight white frothy expectoration every morning. Pulse 96, soft; breath short

on exertion; fauces relaxed and granular, increased by coughing or torpid bowels.

Physical signs.—Right side, wavy breathing; prolonged expiratory murmur; slight crepitation beneath the clavicle and above the scapula, where the breathing was bronchial in character. Left side, wavy breathing with prolonged expiration; tubular respiration at base. Aphonia lasting several months, relieved by creasote inhalations, the application of nitrate of silver to the glottis, and the removal of the uvula.

A sea voyage advised, but patient being able to teach in his school, could not be prevailed on to leave England.

February, 1869.—Sputa copious and muco-purulent; pulse 115; night sweats; weight 9 st. 10 lbs., when first seen 10 st. 4 lbs. Right side, bronchial breathing; bronchophony in subclavian region; slight crepitation in supra-scapular fossa. Left dull, on forced inspiration, at the base. Went to Malta, where he spent three months happily, but made no progress.

August 24th.—Physical signs same but that humid crepitation was noted at the left base, with friction sound below scapula.

Patient now anxious to go a long voyage; dissuaded from it, but went and gained good on voyage, but not on shore at Melbourne, so returned home to die.

CASE 14.—*Cerebral exhaustion, with persistent headache; voyage to Australia, and life at Melbourne very beneficial.*

A tall delicate boy, æt. 17, formerly lazy, made a sudden violent effort to work at high mathematics; in a few weeks rose from the bottom to the top of his class. But headache, at first slight and occasional, became severe and uninterrupted; was obliged to leave school, and rest at home, and in Derbyshire. At the end of four months the headaches were not constant, yet recurred whenever he read, even a novel, for more than half an hour.

A long sea voyage was now advised. An opportunity of

taking a trip to the Mediterranean occurred, and he went to Gibraltar, Malta, Alexandria, and back. When at Alexandria was exposed to the midday sun in an open boat, and had an attack described as "sunstroke," during which consciousness was lost for half an hour, and great drowsiness and torpor followed. Prior to this attack he had felt well, and was almost free from headache, but afterwards the old head pain recurred. On his return to England he had improved in appearance, but there was no gain in power of application to work.

At the end of a year from the first attack a voyage to Melbourne was undertaken. During the voyage, after passing Madeira, he felt perfectly well, had a splendid appetite, gained flesh and strength, and on his arrival in Australia described himself as better than since commencement of his illness. Went up to the gold mines, at Egerton, and to the Murray River. Improvement progressed, and he now describes himself as well, living in the saddle; he can bear great fatigue of body, and can read or write for hours without headache. A medical man, consulted in Melbourne, advises him to spend a second year in the bush, and believes that then he will be able to return safely to England, and work for the bar.

Somewhat analogous to this case, although differing in its result, is the following:

A medical student, anxious to take high honours at the M.B. Examination, worked sixteen hours a day for several weeks, broke down from sleeplessness and persistent headache, followed by disturbed vision, and convulsive muscular twitchings. A year's rest in England was of little benefit, and he could not work. Went to Sydney and back with great benefit. On his return he was able to work cautiously, with the view of taking the M.R.C.S. He was attacked with scarlet fever; on the second day cerebral symptoms set in, and on the fourth day he died comatose.

The cerebral mischief, though controlled by the voyage, was clearly in this case only partially benefited; a further sojourn in the colony might have established the cure.

Case 15.—*Two phthisical brothers benefited by the voyage to Australia and life in the elevated health resorts of the Murray River.*

E. F—, æt. 24, Luton, first seen November, 1871. Had cough for six months; improved at Ventnor in September; breath short; cough and dyspnœa on exertion; scanty sputa; lost a stone in weight, but is now gaining (weight 10 st. 4 lbs.). Pulse 94, soft; tongue pale and patchy.

Physical signs.—Left side flattening, dulness upper third; feeble breathing to base; crackling on deep inspiration in third interspace. Right side normal.

Went to America and Canada for six weeks.

July, 1872.—Overwork drilling with volunteers; increased dyspnœa; weight 10 st. 9 lbs. Physical signs same as last year. Voyage to Australia or South Africa advised.

Got less cough, no expectoration; pulse 90; out shooting; active out-door life suits, warehouse does not; appetite large, drinks two or three pints of beer daily.

December, 2nd.—Never free from cough; weight 10 st. 13 lbs.

Physical signs.—Left, dull feeble; dry crackle on cough upper third front and back. Started for South Africa and was so hearty and vigorous that he was laughed at on the voyage for considering himself an invalid, and feels perfectly well in Australia.

Case 16.—Brother of the above, æt. 24, Luton, farmer, first seen November, 1872. Always delicate; height 5 feet 11 inches; weight 8 st. 7 lbs. Mother's family phthisical. Cough two years, loss of flesh (weighed 9 st. 4 lbs. two months ago); pulse 112; lives in a cottage, low rooms, ten feet square; farm exposed and on the clay; a year ago had pleurisy, never well since.

Physical signs.—Dulness throughout right side, respirations amphoric with crackling above and feeble below. Left side, breathing puerile.

Under alkalies and cod-liver oil improved, and the crepitant signs became more dry.

In December started with his brother in the 'Windsor Castle' with nine other valetudinarian youths. Dangerous storm off the Cape; had dysentery in the tropics and was very ill (with pleurisy) on the voyage. At Melbourne improved, and the improvement was more marked when last heard of on the Murray River Highlands; he was living in the bush, riding much and had gained seven pounds in weight.

Remarks.—These brothers have both gained by the voyage and Australian sojourn. The first may have regained permanently established health. The second was suffering from such advanced disease that arrest only could be anticipated.

CASE 17.—*Recurring bronchitis and asthma; severe in Tasmania and in England, absent in Melbourne.*

Mrs. —, æt. 55, went to Tasmania in 1840 and lived there in health for seven years, but sudden changes of temperature and exposure to keen winds set up several pulmonary, chiefly bronchial and pleuritic, attacks. These became so constant and severe that she was obliged to leave Tasmania for Victoria, where she had no return of pleurisy or bronchitis, but the great unhealthiness of the town told injuriously on the nervous system, and after exposure to summer sun she had sunstroke followed by obscure cerebral symptoms, incapacitating her entirely for two years from the duties of life. The voyage to England restored nerve tone, but on arrival in England she had frequent bronchial attacks complicated with spasmodic asthma, and now a winter never passes, whether spent in London, Bournemouth, or Ryde, without two or three attacks of severe bronchitis. A sea voyage always proves beneficial, and in Melbourne she never had bronchitis or asthma, but was prone to get palpitation and great nervous depression.

Case 18.—*Spasmodic asthma cured during voyage and at Melbourne and in Canada.*

An asthmatic youth suffered so alarmingly from nightly spasmodic attacks that he was ordered to Melbourne. From the time of leaving England till his return (a period of nearly two years) he only had one attack, but on the night of landing at Plymouth he was seized with a bad fit of spasmodic breathing (due possibly to a heavy supper of ham and porter), and, though in better general health, the asthma was very severe.

A voyage to Canada was then taken, and he is now living in health exposed to all the vicissitudes of the Canadian winter, often camping out at night in all weathers.

Case 19.—*Phthisis, second stage; arrest; cure established in Australia and New Zealand.*

An artillary officer, æt. 25, quartered at Gosport; mother phthisical; had cough; hæmoptysis; night sweats; loss of flesh.

Physical signs.—Dulness, with bronchial breathing and crepitation in first and second interspace right side.

Kept in doors at Bath during winter, and in spring joined his regiment and went to New Zealand, where he was actively engaged in the war (obtained promotion for brilliant services). Went to Melbourne, improved health became established; married and is now in England quite well, but there is decided flattening with feeble breathing beneath the right collar-bone.

In this case the amendment was confirmed and established in New Zealand and Australia. Had he remained in England a recurrence of lung disease would probably have occurred.

CASE 20.—*Laryngeal disease and early phthisis, developed in Wellington, New Zealand; voyage to England beneficial; arrest.*

Clerk to Upper House of Representatives, Wellington, New Zealand. After hard work reading aloud long Acts of Parliament in Representative Assembly lost his voice and came here for advice. A tall, thin, flaxen-haired man, æt. 28, of phthisical predisposition, suffering from cough; expectoration occasionally streaked with blood, but mainly from aphonia. First seen in July, 1869. Dr. Morell Mackenzie, who was subsequently consulted, discovered paralysis of left crico-arytenoid muscle. At Ventnor made no progress, and is now delicate in the chest with feeble voice, he is better than when in New Zealand.

The tremendous winds of Wellington ("the Funnel of the World," as it is called), in New Zealand were, by the patient, regarded as greatly causative of his throat affection.

N.B.—A Wellington man can always recognise a fellow townsman when walking in the street by his invariable habit of clutching his hat as he comes to a corner.

The following case, as the evidence of a person with world-wide experience, may be accepted as a contribution of some value.

Chronic hepatic disorder; benefit in Spain, Engadine, Cape Town, and Free State.

A gentleman, never under my professional care, who has travelled the world over in search of health, writes—"What a pity it is that all the dry places of the world (or most of them) should be so disagreeable. The Free State, the desert of Egypt, the central plateau of Spain, are amongst the places that suit me best, but how can one live in such places. Cape Town is the nearest approach to a habitable spot in a dry and warm climate. Next winter I

intend trying Australia, for I will never voluntarily stay in England again after October. To me the most detestable of all English climates is that of Bath and Bristol, where I happen to be often tempted to go to see friends. I never chose the vicinity of fresh water for a residence. Pontrasina, in the Engadine, is the most bracing place I know of. In England I was very liable to bronchial affections, but escaped them almost wholly during a residence of several years in Natal; rheumatism, too, I escaped in Natal."

PRETERNATURAL CAVITIES

IN THE

BRAIN OF THE SANE AND THE INSANE.

BY

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FROM a recent investigation into the comparative frequency of tumours of the brain in the sane and the insane,* in cases that came under my immediate observation in the St. Marylebone Infirmary and the Somerset County Lunatic Asylum, the much greater frequency of an opposite condition, a partial atrophy, in the form of cavities, cysts, and depressions, was so remarkable, especially in the insane, that the examples of this state of the brain appeared to be of sufficient interest to be submitted to the meeting of this learned Society, and the following cases in the sane and the insane, which will be found in the appendix, may serve as an inducement to others to make further investigation.

These appearances may be considered as chronic—the

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result of acute disease, which the person outlived with damaged brain—and as to be expected, most frequently found in the insane. The proportion of chronic diseases of the brain in asylums would, very probably, be found still greater now than thirty years ago, before the building of those large receptacles for the fatuous and bedridden imbeciles, formerly the occupants of chronic wards and infirmaries of workhouses. For the study of morbid anatomy the value of such institutions cannot be overrated, and the admission to them of medical students being legally authorised, great additional sources of most valuable practical knowledge might be made available.

Before comparing these morbid appearances of the brain in the sane and the insane, it is proper to state that of the 1039 post-mortem examinations made in the infirmary, 89 were infants and 236 children under fifteen years, and that in only one of these cases, a boy, No. 950, was there a cavity from an enlarged or dilated blood-vessel in the brain. By deducting the infants and children, and nineteen cases of insanity, 695 infirmary cases remain, 338 males and 357 females, of like age, for comparison with 875 post-mortem examinations made of adults, 502 males and 373 females, in the asylum.

Cavities, cysts, and depressions were found in 68 instances, in 16 males (not including the boy) and 12 females in the infirmary, being equivalent to 47·3 per 1000 in males, and 33·3 in females; and in 26 males, and 14 females in the asylum, being equivalent to 51·7 per 1000 in males, and 37·5 in females, in about 40 per 1000 of the sane, and 47 per 1000 of the insane; whereas tumours of the brain were found in only 18·3 per 1000 of the sane and 16 per 1000 of the insane.

The older writers, Bonetus, and especially Morgagni, have described apoplectic cysts and cicatrices in the brain, attended by hemiplegia of the limbs on the opposite side. Pits and cavities in the brain were found in ten out of fifteen instances by Morgagni on the right side; in two, both on right and left; and in three only on the left side.

In a woman cured of an apoplexy by Brunnerus, five years before her death, there were "three little caverns which had been long formed, lying about the corpus striatum, now grown callous and covered over with a cicatrix, the hemisphere being flaccid, of a dark yellowish colour and shrivelled, as if from an atrophy."

Preternatural cavities have been generally found in the corpus striatum, or optic thalamus, or near one or both of them, and then generally attended with a perforation and laceration of these parts.

Dr. Carswell states that blood effused into the brain may be absorbed and the wound cicatrized, and that soft cerebral substance may also be absorbed. Cruvelhier* relates several instances of the yellow discoloration of the convolutions connected with irregular depressions and atrophy of the grey cerebral matter.

The cerebellum is subject to the same changes as the cerebrum. The French writers believe that the cyst is at last obliterated by the adhesion of its sides. In one case a coagulum had disappeared in five months, in one Serres found a hard coagulum remaining at the end of three years.

Foville† found on the surface of the convolutions in the brain of lunatics, numerous linear depressions and irregular pittings, leaving in the intervals eminences of different sizes. He mentions another state of the brain which is more rare, already noticed by Esquirol, in which there are many small cavities, containing a clear fluid, with clean and white walls resembling porous cheese.

These appearances Dr. Sims considered as traces of t' process employed by nature in arresting or curing white red ramollissement, and are frequently to be met wit dissecting the brains of persons who have in a former pe suffered from apoplexy, paralysis, vertigo, loss of feeling motion.

Dr. Sims states that he had often pointed out t appearances in the brain, which he considered to be traces (

* 'Anatomie Pathologique,' "Maladies du Cerve

† 'Dict. de Méd. et de Chirurg. Pratique,' art.

the arrest, or cure of ramollissement of the brain, in the presence of the medical officers of the St. Marylebone Infirmary, and other professional gentlemen. His cases and observations will be found in the nineteenth volume of the 'Medico-Chirurgical Transactions.' My subsequent experience in the infirmary and in the asylum has confirmed the views I had heard at post-mortem examinations from Dr. Sims. His premature death, from typhus, which prevailed in the infirmary, not long after the publication of his valuable paper, was greatly to be lamented.

In the examples in the appendix, the cysts and cavities were of various sizes, sometimes containing fluid, and lined by a transparent membrane. This adventitious membrane is supposed to secrete the fluid, as serous membranes do in other parts. Abercombie distinctly states, however, that in many such cavities there was no fluid.

Apoplectic cysts, the result of effused blood, are the most numerous, and are to be recognised by the yellow or rusty deposit, owing to peroxide of iron, first pointed out by Dr. Davy, from his examination of some of these cases from the infirmary. The cysts vary much in size and in degree of fulness, from the quantity of clot, or the cysts may be empty, the extravasation being absorbed, the sides of the cysts may have coalesced and a cicatrix, with a dark line in it, have formed (as in Cases 825, male; 832 and 1510 females), terminating with a flattening and atrophy of the parts. Old apoplectic cysts, or cavities in process of healing, were found in 30 out of the 68 cases here recorded in 9 males and 5 females, in the infirmary; and in 11 males and 5 females, in the asylum.

These cysts were usually in the central portions, and more frequently in the right corpus striatum than in any other part of the brain, and accompanied by hemiplegia of the left side. In about one third, these cysts and cavities were found in the cerebral hemispheres, and an unusual quantity of fluid in the same proportion. In the insane, softening of the brain and general paralysis was the result in 4 males and 1 female, softening of the brain only in 2 females.

The brain was unusually firm in 2 males, and the spinal

cord in one of them. In the remaining seven cases the cause of death was from other than cerebral disease. In four from inflammation of the lungs, in two from disease of the heart and dropsy, and in one from peritonitis. Six were in a state of mania, three in melancholia, and seven in dementia. One appeared to be rational, and in two the memory was observed to be good.

Ages.	
Under 40 years .	1 male in infirmary and 2 in asylum.
From 40 to 60 .	2 males and 1 female in infirmary. 4 males and 1 female in asylum.
From 60 and upwards	6 males and 4 females in infirmary. 5 males and 4 females in asylum.
	Total—20 males and 10 females.

Old serous cavities, cysts, and depressions, from loss of cerebral substance, and free from staining, observed in the preceding cases, were found in fourteen instances, the greater number (ten) being in the insane, four only in the infirmary. One of the four was the boy already adverted to, and who evidently died from general dropsy. The other three had cerebral softening, and in the female it was accompanied by gangrene of the cerebellum, No. 772. Two were admitted in a state of stupor. An inequality in the cerebral hemisphere was found in four of the asylum cases. In No. 544, the right hemisphere was devoid of convolutions, a cyst containing half a pint of fluid occupied their place, and there was a difference of four ounces and a half in weight between the hemispheres, in the others the difference was less, the loss of weight in the left hemisphere only; in one case, No. 1181, there was a remarkable amount of cerebral disease, namely, red softening in one part, and an unusual amount of firmness in another, much fluid in cysts in left ventricle, and about six ounces of blood in the spinal canal, the spinal cord firm; skull unusually thick, and brain large in No. 1045 admitted in a moribund state. Serous cysts were found in both cerebral hemispheres in No. 679. These cases terminated in cerebral softening in 4, apoplexy in 1; in pulmonary diseases

in 8, and dropsy in 1; 3 males and 2 females were admitted to the asylum in a state of mania, 2 females in melancholia, and 2 males and 1 female in dementia.

Ages.	
Under 40 years . .	1 male in infirmary. 2 males in asylum.
From 40 to 60 . .	1 male in infirmary. 2 males and 1 female in asylum.
From 60 and upwards	1 male and 1 female in infirmary. 2 males and 3 females in asylum.
	Total—9 males and 5 females.

Small cysts and cavities, arrest or cure of ramollissement.—There were nine examples, five in the infirmary and four in the asylum, of this disease, having the appearance of porous cheese as described by Esquirol, and of *new bread* as described by Sims, and considered by him as a proof of the arrest or cure of ramollissement of the brain. In four of the infirmary cases the mind was affected, either childish or incoherent. Both speech and motion were affected in No. 424, and had been partially restored when death took place from double pneumonia. In No. 742 there was cerebral softening around the new bread appearance, and death from convulsions. Contrary to what is usually observed the disease of the brain and hemiplegia were on the same side in No. 827, and the heart enlarged.

In the asylum, male No. 150 had a fall on his head three years before his admission, he had general paralysis and mania. No. 956 was also in last stage of general paralysis on admission, the new bread appearance was in the white substance of the brain, there was softening of the posterior portion of the cerebral hemisphere as well as of the spinal cord. A female, only æt. 51, had been bedridden from paralysis for several years; she had chronic meningitis, much fluid in the lateral ventricles; the brain was unusually firm, and the new bread appearance in the left optic thalamus. In No. 265, a case of paralysis, the new bread appearance was in each corpus striatum, and softening in the left cerebral hemisphere. These three last cases were all in a state of dementia.

Old cavities and cysts in epileptics were found in thirteen instances, three in the infirmary and ten in the asylum, affording examples of each of the three varieties of cysts, the apoplectic, serous and small porous, already described. Local atrophy to a remarkable extent also existed in some. Sudden death from apoplexy occurred in the three admitted to the infirmary, their mental faculties were not affected, they had all been engaged in their occupations to the last. One of the males, No. 823, had hemiplegia, the limbs small and joints contracted on the left side. His habits uncleanly, he was miserly and had a voracious appetite. There was much fluid contained in a membrane, and atrophy of the right cerebral hemisphere.

Similar peculiarities were observed in a male, No. 83, in the asylum with dementia, and also with atrophy of the right cerebral hemisphere. Six of the ten epileptics were males; and four, females. The ages of the males from 28 to 79, and of the females from 37 to 49 years. The duration had been from childhood in No. 988. There was atrophy of the left cerebral hemisphere, and arrest of development of the limbs on the right side. In Nos. 609 and 83 the fits occurred after an injury to the head. Apoplectic cysts were found in two males. On the outer side of corpus striatum with atrophy of right hemisphere in No. 837, and in No. 609 with softening of the brain, other portions of the brain and spinal cord being unusually firm. Also in No. 566, in the left corpus striatum, the walls of the cysts being considerably contracted, hemiplegia of the right side, of seven years standing. Cysts were found in both corpora striata softening in No. 187, with hemiplegia of the right side a of speech. In one female, No. 246, *an apoplectic cy* softening was found upon the right cerebral hemisphe right olfactory ganglion enlarged, softening and of a colour; an abscess in sphenoidal sinus, skull unusuall and cerebral veins congested.

Serous cysts, which occupied the situation of the al cerebral convolutions were found in two males, on the hemisphere in No. 988, and on the right

Numerous *small cysts or cavities* were found in three females in the right corpus striatum combined with induration, atrophy and an unusual quantity of fluid in the ventricles in No. 429: in the left cerebral hemisphere with cerebral softening, hydrothorax, enlarged heart and granular kidneys in No. 100; and in No. 890, small cavities existed in the centre of each cerebral hemisphere, atheroma of arteries, apoplectic clots in left hemisphere and external to right corpus striatum, inflammation and gangrene of cerebellum in No. 890. As regards the mental state, four males and two females were in a state of mania, which was generally paroxysmal, preceding the fits at irregular intervals. One female was in a state of melancholia, and two males and one female in a state of dementia.

Encysted abscesses in the brain.—According to Abercrombie, one of the various terminations of cerebral inflammation consists of a well defined regular cavity, filled with pus, generally lined by a soft cyst of coagulable lymph, and surrounded by cerebral matter in a healthy state. He also states that tubercular tumours of the brain, having attained considerable size, when cut into, they present the usual whitish coloured or cheesy matter, generally enclosed in a cyst. Nearly analogous to tubercular disease in the brain, appear to be those cases in which albuminous matter in a pure state is deposited in cysts in various parts of the brain or under the membranes. Four cases are recorded by Abercombie, and one by Andral of this nature.

Distinct insulated masses, sometimes indurated, at others softened, have been found enclosed in vascular cysts in the brain, and sometimes large caverns, without mental derangement. In a boy, mentioned by Brodie, there was in the left hemisphere of the brain a cyst about three inches in diameter, containing thick dark-coloured pus, the lower part of it rested upon the petrous portion of the temporal bone, and there was an opening through the cyst, dura mater, and bone, forming a free communication between the abscess and external ear, from which there was a discharge of matter.

The average weight of the brain in the males was 43 oz.

in nine sane, and 45·4 oz. in twenty-five insane, and in the females 41·9 in eight sane and 40·9 oz. in fifteen insane.

The whole of the examples of the various forms of cysts here referred to are set forth, and will be found in the appendix classified as follows:

1st. Old apoplectic cysts, as distinguished by the permanent blood-staining from peroxide of iron. They were the most numerous, and more so in proportion in the sane than in the insane, being 20 per 1000 in the former, and 18·3 per 1000 in the latter.

2nd. Old serous cysts, containing clear fluid and without discoloration, often accompanied by cerebral softening, most frequent in the insane; in the infirmary 4·2, and in the asylum 13 per 1000.

3rd. The small porous cavities, from the arrest and cure of ramollissement. They were less numerous, only nine cases altogether, five in the infirmary and four in the asylum, and mental derangement existed in the infirmary cases as well as in those in the asylum. All the foregoing varieties of cysts were also found in thirteen cases of epilepsy.

4th. Encysted abscess in the brain, of which there were only two examples, both without mental derangement, in the infirmary.

APPENDIX.

Table showing in the 68 cases of cysts, cavities, and depressions in the brain, the sex, age, and civil state, duration of disease, bodily condition on admission, seat of cyst, cause of death, distinguishing the Infirmary and the Asylum cases.

1st.—Old Apoplectic Cysts. 30 cases.

A. 14 *in Infirmary.*

No.	Sex, age, and civil state.	Duration of illness.	Bodily condition on admission.	Situation of cysts, cavities, and depressions.	Cause of death.	Mental state and weight of brain.
1. (453)	M., 38	6 weeks	Paralysis of left leg	Cyst in right corpus striatum	General dropsy	Sane, 44¼ oz.
2. (614)	M., 50, married	10 months	Twitchings of right arm, thin	Cells in left cerebral hemisphere	Pulmonary phthisis	Sane.
3. (825)	M., 59, widower	4 years	Hemiplegia right side	Cicatrices and cells in optic thalami and corpora striata	Ditto	Sane, 44¾ oz.
4. (767)	M., 63	3 years	Lower limbs contracted	Cysts in left lobe of cerebellum and softening	Ditto and pneumonia	Clear mind, 45 oz.
5. (291)	M., 67	Many years	Hemiplegia left side	Cyst in right corpus striatum	Pneumonia of right	Sane.
6. (826)	M., 73	No history	Ditto	Ditto, and also in right lobe of cerebellum	Double pneumonia	Sane, 36 oz.
7. (161)	M., 78	4 days, insensible	Ditto	Ditto in right corpus striatum, much fluid in ventricles	Pulmonary apoplexy	?

8. (114)	M., 80	5 days, insensible	Comatose	Ditto in left corpus striatum, softening, ventricles distended with fluid	Pericarditis	?
9. (649)	M., 87	No history	Bad	Cysts in left corpus striatum	Pulmonary phthisis and pneumonia	?
10. (809)	F., 50	6 days, insensible	Hemiplegia left side, emaciated	Depression on left hemisphere, and softening and clots of blood	Cerebral softening	Sane
11. (830)	F., 64	8 years	Ditto right side	Cyst in left corpus striatum, brain unusually firm	Atrophy of kidneys (5½)	Sane, 44½ oz.
12. (379)	F., 68	Many years	Ditto left side, erysip. and gangrene of great toes	Cysts in left cerebral hemisphere, convulsions	Pneumonia, left.	?
13. (832)	F., 69	2 years	Ditto right side, comatose	Cicatrix in left corpus striatum, much fluid on surface	Atrophy of kidneys (4)	? 38½ oz.
14. (221)	F., 71	No history	Bad, bronchitis, bedridden	Cavity in right hemisphere, much fluid in ventricles	Redness, &c., in bronchial tubes; heart small (3½ oz.)	?

B. 16 *in Asylum, first* 9 *of them terminating in General Paralysis.*

15. (192)	M., 28, single, labourer	6 months, memory lost	Paraplegia lower limbs, &c., convulsed	Cyst, and red softening of right corpus striatum and spinal cord	Pulmonary phthisis and pneumonia; right lung	Dementia, 44¼ oz.
16. (1207)	M., 33, married, miller	19 months, violent	Hemiplegia right side, tongue paralysed	Depression and 3 oz. loss on left hemisphere, central softening also of cord, fluid in ventricles	Cerebral and spinal softening	Mania, 49½ oz.

No	Sex, age, and civil state.	Duration of illness.	Bodily condition on admission	Situation of cysts cavities, and depressions.	Cause of death	Mental state and weight of brain.
17. (144)	M., 45, married, wanderer	27 months, fancied riches, noisy	General paralysis, apoplexy	Cyst in left corpus striatum, brain and cord unusually firm, fluid in spinal canal	Pneumonia right lung; heart large (13 oz.)	Mania, 46½ oz.
18. (811)	M., 47, married, labourer	13 months	Ditto and hemiplegia left side, became convulsed, and speechless	About 4 oz. of bloody fluid in cyst on left hemisphere (1¾ oz. loss), cord soft and white	Double pneumonia	Dementia, 40¼ oz.
19. (1006)	M., 51, single, labourer	6 weeks, intemperate, violent	Bad, refused food, general paralysis	Large cyst full of blood on left cerebral hemisphere, convolutions flattened, loss 1 oz., septum and fornix softened, spinal cord also softened	Congestion of blood in lower lobes of lungs; heart large, 13 oz.; kidneys large (12)	Memory lost, Dementia, 41¼ oz.
[illegible] (972)	M., 60, married, innkeeper	16 months, suicidal	Bad, general paralysis	Small cavity in right corpus striatum, chronic meningitis, brain unusually firm and tough	Lungs congested, heart large (16½); stomach large (7)	Talks rationally, melancholia, 48 oz.
[illegible] (1035)	F., 40, wife of labourer	20 months	Helpless, hemiplegia left side, general paralysis	Cyst in right corpus striatum, veins congested, chronic meningitis, softening of sides and centre of cord	Heart flabby, 12 oz.	Memory good, dementia, 43 oz.
[illegible]	F., 62, married, 17 children	9 months, dangerous to self and others	Bad, convulsions, passed excretions involuntarily	Small cysts in right hemisphere, red softening of left corpus striatum and optic thalamus, exudation corpuscles, cord natural	Cerebral softening; atheroma of arteries	Melancholia, 42 oz.
[illegible] (612)	F., 60, married	[illegible] months, violent	Hemiplegia left side, helpless, can only swallow liquids	Cyst and softening of right hemisphere externally, fluid in ventricles, skull thick, meningitis, cord natural	Cerebral softening	Mania, 40½ oz.

(745)	M., 45, married, quarryman	2 years, from an injury to head	Bad, intemperate habits, no paralysis	Veins congested, two depressions on outer side left hemisphere, brain around indurated, 2 oz. less, spinal cord large ($1\frac{1}{2}$ oz.)	Acute peritonitis; recent lymph and fluid in peritoneum	Dementia, $47\frac{1}{4}$ oz.
5. (734)	M., 65, married, servant	9 years, intemperate habits	Hemiplegia left side, articulation affected, unable to dress himself	Cyst, and softening around size of a walnut in centre of right hemisphere, 4 oz. fluid in ventricles, cancelli of skull red	Cardiac dropsy; heart very large, $19\frac{1}{4}$ oz.; atheroma of arteries	Memory good, melancholia, $47\frac{3}{4}$ oz.
26. (938)	M., 65, married, postman	4 months	Hemiplegia left side, unable to swallow solids	Cyst, and softening of right corpus striatum, atheroma of arteries	Pleuro-pneumonia; softening of spinal cord	Dementia, $37\frac{1}{2}$ oz.
27. (878)	M., 66, single, pauper	2 months, dangerous	Hemiplegia left side, erysipelas, coma	Cyst in right corpus striatum, meningitis, chronic atheroma of arteries	Pleuro-pneumonia; heart large, 13 oz.	Mania, $47\frac{1}{4}$ oz.
28. (441)	M., 67. married, mason	5 years, dangerous to self and others	Hemiplegia left side, very helpless	Cyst and remains of clot in right corpus striatum, atheroma of arteries, clot in upper part of spinal cord	Pneumonia right lung (46); left (16); heart large, $16\frac{1}{4}$	Dementia, $45\frac{3}{4}$ oz.
29. (464)	F., 66, widow	1 year, filthy	Hemiplegia left side, unable to stand, dyspnœa	Small cysts on hemispheres, cicatrix in left optic thalamus, a depression in right corpus striatum, spinal canal full of fluid, cord soft	Fluid in right pleura; heart large, $15\frac{1}{2}$ oz.; kidneys small	Dementia, $40\frac{3}{4}$ oz.
30. (1510)	F., 79, single	7 years, restless	Bad, diarrhœa, no paralysis	Cyst and cicatrix near right corpus striatum, much fluid in ventricles, interior soft	Broncho-pneumonia	Senile dementia, 37 oz.

2nd.—Old Serous Cysts. 14 cases.

c. 4 in *Infirmary*.

No.	Sex, age and civil state.	Duration of illness.	Bodily condition on admission	Situation of cysts, cavities, and depressions	Cause of death.	Mental state and weight of brain
[illegible]	[illegible]	No history	Brought for interment	A cavity at vertex, and dilated blood-vessel passing into sinus	General dropsy; much fluid in chest and abdomen	?
[illegible]	[illegible]	[illegible]	Hemiplegia left side, chronic bronchitis	Cyst in left corpus striatum, clear fluid in it and much in ventricles, portion of right hemisphere pulpy	Cerebral softening	Sane, 44½ oz
[illegible]	[illegible]	[illegible]	Stupor, right leg contracted, broncho-pneumonia	Cyst in each corpus striatum, softening in right hemisphere	Cerebral softening and pneumonia right lung; kidneys small (5¼)	? 45½ oz.
[illegible]	[illegible]	[illegible]	Hemiplegia some years, gangrene cerebellum	A depression and softening of right corpus striatum and right hemisphere, also of cerebellum	Cerebral softening, kidneys small	? 42 oz

d. 10 in *Asylum*.

No.	Sex, age and civil state.	Duration of illness.	Bodily condition on admission	Situation of cysts, cavities, and depressions	Cause of death.	Mental state and weight of brain
[illegible]	[illegible]	[illegible]	Indifferent, pupils dilated, [illegible] thigh, abscess, destructive, opisthotonos	Cyst on right hemisphere contained 8 oz., loss in weight of 4½ oz., no convolutions, spinal canal full of fluid, cord soft	Double pleuro-pneumonia; myelitis	Mania and general paralysis, son of Louis Philippe, 36 oz.

[illegible]	[illegible]	[illegible] months, violent	Moribund, pupils contracted, convulsions, died 19 hours after admission	Cysts in left ventricle and fornix, lobe pale, firm, red softening, right corpus striatum and crus cerebri, blood in spinal canal	Softening of brain; left lobe lost 4½; chronic inflammation spinal cord	Dementia, brain large, 51½ oz.
[illegible]	[illegible], [illegible]le, tailor	9 months, quarrelsome, affections changed	Indifferent, had a fit from which he never recovered 7 months after admission	Cyst in right hemisphere, parts around soft, other parts unusually firm, cord natural, veins congested	Apoplexy	Memory bad, mania, 39¾ oz.
[illegible](079)	M., 49, married, miller	8 months, affections changed	Bad, emaciated from pulmonary phthisis, noisy, destructive	Two small cysts on surface of hemispheres, skull thick, opacity of arachnoid, fluid on surface, cord firm	Tuberculous cavity and bronchitis	Memory bad, mania, 45¼ oz.
[illegible]7)	M., 67, married, brickmaker	8 years, after fever, violent	Bad, anasarca, paralysis bladder, phlegmonous erysipelas right arm	Two small cysts in left corpus striatum, chronic meningitis, fluid on surface	Lungs congested; heart, 12 oz.; valves thickened; atheroma of aorta.	Affectionate dementia, brain small, 36¼ oz.
40. (1181)	M., 68, widower, lamplighter	2½ years, injury to head, dangerous	Paralysed, suffered from pneumonia of left lung for five weeks before his death	A depression on outer side of left corpus striatum, size of threepenny piece, left hemisphere lost 1½ oz., brain large and firm	Left lung hepatised (30); right (17)	Mania, 52¾ oz.
41. (469)	F., 51, widow	Not ascertained, 8 months in asylum, suicidal	General paralysis, articulation indistinct, could scarcely walk	A small cavity, dilated vessel in left hemisphere, atrophy, upper part of spinal cord softened, spiculæ of bone in cauda equina	Tubercles in right lung (23); left (40); from pneumonia	Melancholia, memory good, brain only 34¼ oz.
42. (54)	F., 64, widow	4½ years, excited and noisy	Indifferent, erysipelas, cardiac dropsy	Cyst in right corpus striatum, left hemisphere 1 oz. heaviest, fluid in spinal canal	Two pints fluid in pleura; heart 14, valves thick; fluid in abdomen; ulcer in stomach.	Mania, 48¾ oz.

No.		Sex, age, and civil state.	Duration of illness.	Bodily condition on admission.	Situation of cysts, cavities, and depressions.	Cause of death.	Mental state and weight of brain.
43.	(10)	F., 68, married, 7 children	22 years, cheerful, industrious	Good, for 3 years before death asthmatic	A cavity 4½ by ½ inch in right hemisphere, loss of weight of 2½ oz., cord natural	Influenza; bronchitis	Chronic mania, 42½ oz.
44.	(1527)	F., 85, widow	1½ year, troublesome in workhouse	Bad, paralysed, unable to walk or feed himself, bed-sores on hips	Depression on right corpus striatum, ventricles distended with fluid, firm brain, cord natural	Emphysema of lungs; the right congested	Memory lost, senile dementia, 39½ oz.
		3rd.—Small Cysts and Cavities. Arrest or cure of Ramollissement. 9 cases.					
		E. 5 *in Infirmary*.					
45.	(424)	M., 45	4 months, loss of speech, wh. he recovd.	Hemiplegia right side, limbs partially restored	Small cyst in left corpus striatum, and smaller in parts around, and unusual firmness	Double pneumonia	Childish.
46.	(742)	M., 70	5 weeks, convulsions	Bad, acute headache, 3 weeks at home	Cellular state (new bread), and softening post. cerebral hemisphere, arachnitis	Congested lungs; arachnitis and cerebral softening	Delirium, 45¾ oz.
47.	(827)	F., 46, pauper in workhouse	Several years, convulsed	Bedridden, hemiplegia left side, disease same side as brain	Small cysts in left corpus striatum and near to it, atrophy and fluid	Emphysema; heart large (13)	Dementia, 40 oz.
48.	(62)	F., 52, pauper	13 months, convulsions	Paralysis and contraction left leg	Numerous small cysts lined by membrane in both hemispheres, ventricles full of blood	Cerebral disease	Dementia, 41¾ oz.
49.		F., 72	No history previous, 2 wks. in infirm.	Unconscious, face flushed, head hot	Several small cavities in hemispheres, parts around firm, vessels congested with blood	Apoplexy; bronchitis	? 42 oz.

F. 4 *in Asylum.*

50.	(150)	M., 46, married, coachman	6 months, injury from a fall, violent	General paralysis, convulsed before death	Small cysts in each optic thalamus and right corpus striatum, with softening of these and left crus cerebri, and left lobe of cerebellum, also softening of lower portion of spinal cord	Softening of brain and spinal cord; double pneumonia; enlarged heart and kidneys	Affections changed, memory bad, mania, 44 oz.
51.	(956)	M., 76, married, labourer	1 month in asylum, reported dangerous at home	Bedridden, restless, had to be fed, general paralysis	Small cysts in "centrum ovale of Vicq d'Azyr," softening of posterior portion of right hemisphere, and pus-globules found in it, spinal cord softened	Cerebral and spinal cord softened	Senile dementia, brain large, 52¼ oz.
52.	(640)	F., 51, single, servant, 1st attack	Several years, in workhouse	Bedridden, paralysis, bed-sores, could only swallow fluids, emaciated	Spongy, new bread state of left optic thalamus, septum transparent, ventricles distended with fluid, meningitis, spinal cord natural	Pneumonia right lung	Dementia, 41¾ oz.
53.	(265)	F., 77, pauper	21 months, troublesome in workhouse	Paralysis, filthy habits, for last week difficulty in deglutition	Small cavities in each corpus striatum, posterior portion of left hemisphere wasted and softened, spinal cord rather soft	Softening of brain and red pneumonia	Dementia, 40¼ oz.

CYSTS IN EPILEPTICS. 13 Cases, affording Examples of the three foregoing Varieties of Cysts.

G. 3 *in Infirmary.*

54.	(795)	M., 32, painter	Several years, frequently in infirmary for epilepsy	Full habit, sudden death in a privy, coroner's inquest	Cellular cavity in left hemisphere, veins congested, a large clot on posterior part of right hemisphere	Cerebral apoplexy; heart, 12 oz.; kidneys small	Sane, worked at his trade, brain large, 50¼ oz.

No.	Sex, age, and civil state.	Duration of illness.	Bodily condition on admission.	Situation of cysts, cavities, and depressions.	Cause of death.	Mental state and weight of brain.
55. (823)	M., 47, single, linendraper	Congenital, brought by police, fell down in a fit	Insensible, died 2nd day, mouth drawn, hemiplegia left side from birth, limbs contracted and small	No cerebral convolutions, encysted fluid on right side hemisphere 9¾, left 18½, natural	Fluid on right hemisphere; epilepsy	Sane, clever in business, 33¾ oz.
56. (811)	F., 50, laundress	Several years, epileptic, flushings and giddiness	Apoplectic coma 2 days before death, subject to headache, hemiplegia right side	Cyst in left corpus striatum, a large clot in right corpus striatum and ventricle, a tumor of dura mater, and cavity on left hemisphere	Cerebral apoplexy	Sane, worked at her business to the last, 42¼ oz.
			H 10 *in Asylum.*			
57. (837)	M., 28, single, leather-dresser	2 years, had 3 fits in a month, dangerous to others	Indifferent, sank into a semi-comatose state before death, in a fit	A cyst size of a four-penny piece on outer side right corpus striatum, loss of 1½ oz., congestion of blood in theca vertebralis	Cerebral apoplexy; veins congested	Mania, 51 oz.
58. (988)	M., 29, pauper	From infancy, dangerous to self and others	Hemiplegia right side from arrest of development, limbs contracted and shorter than on left side	Fluid in cyst instead of anterior portion of left hemisphere, brain sent to Pathological Society	Pulmonary phthisis; 33 fits by day and 5 by night in one year	Mania, 49 oz.
59. (187)	M., 34, married, shoemaker	17 months, aft. apoplexy, 5 weeks in St. Bartholomew's Hosp.	Hemiplegia right side, loss of speech, unable to swallow, has to be fed, mouth open, could protrude his tongue after being electrified	Apoplectic cysts and softening of both corpora striata and walls of 4th ventricle, left hemisphere 1 oz. less than right, lower portion of spinal cord soft	Cerebral softening and double pneumonia.	Understanding and memory good, kind and affectionate, 48¾ oz.

60. (609)	M., 44, widower, leather-dresser	12 months, injury to head, noisy at night, excitable	Bad, intemperate habits, wanders about at night, spasmodic twitchings of left shoulder	Small apoplectic cyst in anterior portion of right corpus striatum, much fluid in ventricles, brain and cord firm, skull thick	Convulsions and paralysis	Memory bad, mania, 46½ oz.
61. (83)	M., 48	29 years, from an injury to the head	Filthy habits, limbs on left side smaller by one inch in circumference than those on the right	Right cerebral convolutions absent, fluid in cyst in place of them, 6 oz. less than the right, spinal cord natural	Convulsions at last; pleuro-pneumonia left lung	Dementia, 41¼ oz.
62. (566)	M., 79, widower, tailor	2½ years, epileptic, suicidal, at Hanwell	Hemiplegia right side, 7 years, had partially recovered use of limbs	Contracted cyst, ¾ inch long, 2 lines broad, in lower part of left corpus striatum, membranes thickened and adherent	Lungs congested with blood; heart large and flabby, 15 oz.	Memory good, delusions, mania, 43 oz.
63. (246)	F., 37, widow, Infant-school teacher	5 years, has suicidal propensities	Indifferent, vertigo, want of sleep, 2 or 3 fits every day, none by night	Apoplectic cyst and softening in left hemisphere, crista galli sound, abscess in left spheroidal sinus, skull unusually thick	Convulsions; cerebral softening; lungs congested	Melancholia, 42½ oz.
64. (1100)	F., 47, married, weaver	1 year, has had several attacks of puerperal mania	Indifferent, fits irregular, one month 11, next perhaps none, then 5, or 3 in a month, had dropsy and dyspnœa finally	Small cysts in left corpus striatum and in the hemisphere, softening around, chronic meningitis, brain small, spinal cord soft at centre	1½ pint fluid in pleura; lungs congested; heart large, 14 oz.; kidneys small and granular (2¾)	Mania, 36 oz.
65. (429)	F., 48, single, governess	8 months, fits, rapid in succession	Bad, filthy habits, requires to be fed, emaciated, 67 lbs.	Numerous small cysts behind left corpus striatum, in centrum ovale, brain pale, 1 oz. fluid in ventricles, cartilaginous plates size of four-penny piece on spinal arachnoid	Convulsions; cerebritis	Dementia, brain indurated, 37¾ oz.

No.	Sex, age, and civil state.	Duration of illness.	Bodily condition on admission.	Situation of cysts, cavities, and depressions.	Cause of death.	Mental state and weight of brain.
66. (890)	F., 49, married	4 years, fits frequent, died 6 weeks after admission	Filthy habits, bedridden, bed-sores, maniacal paroxysms	Minute cavities in each hemisphere, a small clot in right corpus striatum and in posterior portion of left hemisphere, recent lymph on cerebellum, greenish and fœtid odour, atheroma of arteries	Cerebritis	Mania, brain firm, 47½ oz.
9th.—Encysted Abscess in the Brain. 2 Females in the Infirmary.						
[illegible]	F., 19, single	3 months, temporary relief from cupping, pupils dilated, countenance pale	In last stage of pulmonary phthisis, subject to hæmoptysis after any exertion (11 yrs.), catamenia absent 4 months, intense headache, increased by erect posture, sickness and vomiting, no cough, dull sound and fluttering in right subclavian region	Dryness and unctuosity of the arachnoid, flattening of convolutions, veins distended, an abscess size of a shilling on back part of left hemisphere and one on front, another in left corpus striatum, and pus in ventricle, a fourth abscess in right hemisphere, each lined by a membrane, pus on cerebellum	No convulsions; four abscesses in brain; examination of head only permitted	Mental faculties entire to the last
[illegible]	[illegible], 61, [illegible]	3 months, two days before admission she fell in the street paralysed	Speechless, left arm swollen and wrist contracted, right arm rigid, deglutition difficult	An abscess size of a walnut in right hemisphere behind, and a smaller one in left corpus striatum, 2¼ oz. fluid in ventricles, four bony spiculæ on falx cerebri	Paralysis; bronchitis	Unconscious, 44¼ oz.

ON THE

INFECTIVE PRODUCT OF ACUTE INFLAMMATION.

BY

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The following paper contains the results of researches made for the most part during the earlier months of 1872, for the purpose of elucidating the pathology of acute secondary inflammation. It forms part of the results of an inquiry undertaken at the expense of the Medical Department of the Privy Council, and is now presented to the Society with the sanction of the head of that department, Mr. Simon.

The purpose of the paper is to show, 1st, that in all acute suppurative inflammations the exudation liquids exhibit, if they are introduced either into the circulating blood, the [illegible] cavities, or the cellular tissue, poisonous or infective [illegible]; 2ndly, that these properties manifest themselves [illegible] the production of increase of [illegible] to secondary inflammation;

3rdly, that the increase of temperature is the direct result of the presence of the poisonous or infective agent in the circulating blood; 4thly, that the secondary inflammations are of two kinds, which may be distinguished from each other by the terms acute and chronic, and that the acute secondary inflammations differ from the chronic not merely as regards duration, but as regards their anatomical characters.

The author arrives at no conclusion as to the nature of the substance to which the pyrogenic property of exudation liquids is due. In the acute process, attended with pyrexia and the development of secondary inflammations of great intensity, there is evidence to show that we have to do with septic decomposition; not only in the fact that bacteria are present, both at the foci of inflammation and in the blood, but also in the perfect identity of the symptoms with those of septicæmia. It is not, however, as yet proved that in chronic infection the irritant is of the same nature; for, although there are all gradations between septicæmic infections of the most rapid development, and slow processes of tuberculosis, this gradation in itself affords no evidence that the two are dependent on the same material cause.

The paper is divided into three parts:

In the first the nature of the process of inflammation is shortly discussed with a view to the consideration of the manner in which a primary inflammation gives rise, on the one hand, to general constitutional disturbance, *i. e.* fever; and, on the other, to the establishment of new foci of inflammation in other parts remote from the original seat of irritation or injury.

In the second part an account is given of certain preliminary inquiries conducted by the author in the years 1867–68.

In the third part the experiments on which our present conclusions are founded are described and those conclusions stated in detail.

Part. I.—Introduction.

When a living tissue is injured mechanically or chemically without being destroyed, it becomes the seat of a succession of changes, which are the products of the disorder of the vital functions of the injured parts. These changes collectively receive the name Inflammation. The term comprehends, therefore, an assemblage of phenomena, held in relation with each other by the circumstance that they are all effects of the same injurious agency, and that they all form parts of one process, of which the various stages follow each other in more or less orderly succession.

It is not needful, in introducing the present inquiry, to describe the process of inflammation, for the question which concerns us has relation rather with the agent by which the phenomena are produced than with the manifestations of its action. It will be sufficient to remind the reader that the phenomena are of two kinds—those which depend on changes in the structural elements of the tissues, and those which have their seat in the blood-vessels; and that the latter, again, admit of a very obvious division, into those which concern the blood-vessels as muscular tubes (the contractile elements of which are under the immediate control of the vaso-motor nervous system), and those which relate to their function as mere conduits for the conveyance and distribution of blood.

If we compare these two orders of phenomena (the vascular and the textural effects of injury) with reference to their importance as characteristics of the process of inflammation, we shall assign the first place to the former, leaving the latter in subordinate and consequential relation to it; for although we know that a living part cannot be injured without the elements of its tissue undergoing those germinative changes of which the production and multiplication of young cells is the result, yet observation teaches us that this cell germination is never the first link in the chain of

effects of which the reaction of a living tissue against an injury consists.

The first local effect which an injury produces in a living part is vascular; it manifests itself as above indicated in two directions simultaneously. On the one hand, the state of contraction (or tonus) of the smallest arteries is altered or modified within and around the seat of injury in such a way as to determine increased supply of blood to the injured part; on the other, the walls of the capillaries undergo imperfectly understood changes, by virtue of which the liquor sanguinis (plasma) and corpuscles sweat or squeeze out into the lymphatic spaces in which the elements of the tissues lie.

This sweating out of the liquor sanguinis we call exudation, using the term in its original and etymological sense, as distinguished from that which has been more recently given to it. Shortly after the introduction of the microscope as an instrument of pathological research, the microscopical study of the cellular products of inflammation so completely engrossed attention that by many writers the process or injury by which they were produced was altogether forgotten or left out of sight. So much so is this the case that in some of the best known text-books of twenty years ago the word exudation is used as name for certain sorts of liquids (purulent, tuberculous, &c.), or for the cells which were then supposed to characterise them. On this account it is necessary to state that by exudation we mean the act of exudation, and not the liquid resulting from that act.

The question of the relation between this act and the textural changes which are associated with it in the process of inflammation may, in accordance with what has preceded, be thus stated:—The tissue elements begin to germinate because they are stimulated, but the exciting action may be either identical with that by which the process is originated, or a secondary consequence of that action; in other words, the tissue may be acted on directly by the mechanical or chemical cause of the inflammation, or mediately by the flood of exuded liquor sanguinis with which it is irrigated.

I have elsewhere stated* why it appears to me probable that in most inflammations the excitation of the tissues is indirect or secondary. This inference is mainly founded on the consideration that whereas we know of no instance of germination of tissue without exudation preceding or accompanying it, it can be proved by experiment that the introduction of exudation liquid into a healthy living part at once determines germinative changes. Admitting the facts, the proof is still incomplete, for it is not denied that there may be examples of textural germination occurring under the immediate influence of mechanical or chemical irritation without any antecedent exudation. If such instances exist they are at all events exceptional.

The notion that the liquid which is exuded in an injured part, as the immediate result of the injury, is itself the exciting agent by which the ulterior and more obvious changes are determined, appears at first sight to stand in very close relationship with the subject of the present inquiry; for if we say that all inflammatory liquids, even those of the most simple inflammations, are endowed with the property of irritating or exciting the elements of the tissues with which they come into contact, it seems very much the same as if we were to say that all inflammations are infective or contagious. If the word infective were to be understood as merely signifying the injurious action of a diseased product on healthy tissue, infectiveness would certainly be an attribute of every phlegmonous liquid. The difference in this respect between one inflammation and another would be merely a question of degree. But before accepting such a view of the subject, which, however logical it may appear, carries with it something which is opposed to experience, we must carefully consider whether the definition above referred to truly expresses the sense in which the word infective is intended to be used.

In the current language of the bedside it is usual to speak of inflammations being "healthy or unhealthy," and to

* "The Process of Inflammation." 'Holmes' System of Surgery,' vol. v, p. 786.

distinguish between *pus bonum et laudabile* and other sorts of pus to which these favorable adjectives cannot be applied. The characters by which the good or bad quality of a purulent liquid, or the healthy or unhealthy conditions of a wound or granulating surface, may be distinguished are plainly set down in practical works, and thoroughly appreciated by every practical surgeon; and it is also well understood that unhealthy inflammations are not only distinguished by their objective characters, but also by the property which they have to produce general disorder of the health of the patient, and in their turn to be injuriously affected in their progress by the disorder they have themselves produced. The constitutional state which is associated in this twofold way with inflammation as its product and its cause, exhibits variations which do not always correspond either with the extent or even with the intensity of the local process which it accompanies, and receives names which express that variation. It may be admitted that in all cases inflammation, if of considerable extent, is attended with fever; but there is a marked difference between the slight febrile disturbance which accompanies healthy suppuration and the degrees of general disorder which are expressed by such terms as "irritative fever," "pyæmia," or "septicæmia." These three pass into each other by insensible gradations, but are separated from health by a line which has become much more easy to define practically at the bedside since the thermometer was introduced into every-day use as an aid to clinical observation.

To distinguish those inflammations which produce grave constitutional disturbance and secondary lesions by an expressive word we call them *infecting*. In using this word we are guided more or less by the same criterion as those on which the surgeon bases the practical distinctions above referred to. An inflammation which is more or less exactly limited in duration and extent by the original limits of the injury which has caused it, may, with scientific precision, be designated a simple or normal inflammation. An inflammation which spreads and endures beyond the direct and

primary operation of its cause, which induces similar inflammations in other parts, and disorders the vital functions of the whole body, has in it something beyond the effects of the injury, and may properly be termed *infecting*. Thus, infectiveness is marked by two sets of characteristics, one relating to what occurs at the original seat of inflammation, the other to the induced effects which manifest themselves elsewhere. Of the two groups of phenomena it is obvious that those which are removed from the seat of action claim most attention, for they afford evidence that material must have been discharged from the original focus, either by the absorbents or the veins, into the circulation. They consist partly in the springing up of new foci of inflammation along the course of the infected channels (the anatomical distribution of which secondary foci always distinctly indicates the source from which they have originated), partly in the occurrence of changes in the physical and organoleptic characters of the blood itself (not as yet investigated), of such a nature as to show that it is impregnated with the infective poison.

In the extended series of experiments made by the author in 1867 and 1868, one branch of the question of phlegmonous infection was worked out with some completeness. It was then found that when in the lower animals local inflammations are produced either in the skin or peritoneum by the introduction of irritant substances, two distinct sets of consequences manifest themselves, viz.:—(1) a chronic disease exhibiting in all respects the anatomical characters of tuberculosis, and consisting essentially in the overgrowth of certain tissues then designated as lymphatic or adenoid, and shown to be in close relation with the lymphatic system; and (2) an acute disease presenting the leading features of pyæmia, attended with the formation of metastatic abscesses, and as a rule terminating fatally either very rapidly by intense peritonitis without any other appreciable lesion, or more slowly by the formation of infective abscesses and nodules, associated with inflammation not only of the peritoneum, but of other serous cavities.

These two morbid processes, the chronic and the acute, are both infective in the sense in which the word has been defined above. Both may apparently spring from an infection derived from the same source, *i.e.* from the same primary infective focus, and may progress in the same animal at the same time. These facts, however, afford no sufficient ground for regarding the two processes as identical, for, notwithstanding the closeness of their relation to each other, the differences cannot be overlooked.

The first reason for considering the two processes as different is to be found in the fact that, to a certain extent either may be produced independently of the other at the will of the experimenter. In a former paper it was shown that if an extremely small quantity of material from an enlarged and indurated gland of a tuberculized—*i. e.* chronically infected—guinea-pig is injected into the pleura of another animal of the same species it may happen that no appreciable constitutional disturbance is produced at the time, and so little local effect that if the animal is killed within two or three weeks after the inoculation no trace of lesion can be detected in the pleura or elsewhere. Eventually, however, the animal becomes tuberculous, the process beginning by the enlargement of the minute lymphatic masses of the pleural serosa. More conclusive ground for regarding the two processes as pathologically different is to be found in the difference of their development. The acute affection attains its acme shortly after the infection, and associates itself in its progress with the primary inflammation produced at the seat of injury by the introduction of the infective agent. The chronic affection, when its phenomena are not complicated and modified by being mixed up with those of the acute, progresses so gradually that the appearance of the lesions seems to be preceded by a period of latency, during which the seed, so to speak, fails to germinate. Not that it can be supposed that the infective material is really latent, but that the earliest changes are so inconsiderable that they cannot be readily discovered.

The investigations which I have now to submit to the

Society enable me to state, more clearly than before was possible, what is the true relation between these processes. The experiments differ from those referred to above in two important respects. In the first place, the material employed for exciting the primary inflammation was, as a rule, a product of an acute process; and secondly (what is of much greater importance), the quantity used for each insertion was ten or twenty times as large. Consequently in all cases the results obtained were, if the expression may be allowed, pyæmic rather than tuberculous. The animals were affected at once, suffered as a rule from well marked constitutional disturbance from the first, and died with or of intense inflammation in the serous cavities. The comparison of the lesions observed in these animals with those above referred to shows that in the acute cases, provided they live long enough, disseminated nodules are to be found in the serous membranes and in the substance of the abdominal and thoracic viscera, which soon assume the character of abscesses, and have the same distribution as the miliary nodules of the chronic process; or at all events a very similar one. They differ, however, alike in their prevalent characters and aspect, in their size, in their duration, and in the changes which they undergo.

Instead of slowly growing, as if they were tumours, lasting for months in the vascular state, and eventually dying at their centres by a process of caseation as gradual as their growth, they enlarge rapidly, are soft from the first, and eventually assume the character of absc[illegible] We must, however, guard against the assumption th[illegible] any original structural difference between th[illegible] and suppurating nodule and the slowly [illegible] granulation, for both originate by overgrow[illegible] elements—in other words, by germination.[illegible] between them is not one of origin or even [illegible] of duration and development; one grows as [illegible]

* We use the word in the same [illegible] term "proliferation" is used by [illegible]

of the tissue from which it springs, until by the redundant multiplication of its closely packed elements, its central parts lose their blood supply; whereas the other becomes itself the seat of an acute inflammatory process. The conversion of a granulation into an abscess is, I think, perfectly parallel to the change which the granulating surface of a wound undergoes under the same conditions, *i. e.* when it assumes the unhealthy character. Just as the newly formed vascular granulation softens and breaks down by a process of inflammation determined by the change which has taken place in the constitutional state of the patient, so we may readily understand that when the inoculated animal becomes the subject of infective fever, the interstitial indurations and granulations which would otherwise have retained their vascularity, firmness, and transparency for many months, may become acutely inflamed and become the seat of abscesses.* In like manner in phthisis pulmonalis in man the local condition of the diseased organ not merely determines the constitutional state, but is afterwards affected by it; so that the setting up of irritative fever from whatever cause determines the softening of parts which would otherwise have remained in a state of chronic induration.

In my introductory report on the intimate pathology of contagion, published in 1870, I brought together the general grounds which exist for the belief that the *materies morbi,* or contagium, of contagious diseases in general, does not consist of substances chemically dissolved in the morbific liquid. The direct evidence in favour of the proposition was to be obtained from experiments which proved that in the case of some of the best known examples of infective action the toxic agent could be shown to be incapable of diffusion, and therefore in the strict sense insoluble. It was further argued, in the second part of the same paper, that if infective agen[illegible] are particulate they are probably comprised in that group [illegible] bodies to which I then applied the term microzymes—rec[illegible] nising their identity with the *zooglæa* of Cohn, the [illegible]

* It has not yet been proved experimentally that it is

of Hallier, and the various forms described by other authors under the terms *bacterium* and *vibrio.*

With reference to these organisms, two entirely new and most important facts have been demonstrated by the observations to be now recorded.* It has been discovered (1) that in all acute infective inflammations microzymes abound in the exudation liquids; and (2) that the same forms are also to be found in the blood of the infected animals, their presence being a constant accompaniment of all acute infective suppurations.

The more detailed statement of these facts will be found in the account to be immediately given of the experiments. It will, however, be convenient, in order to facilitate the understanding of that description, to explain here certain words which we have been in the habit of using to designate the varieties of form which are most frequently met with according to their grouping and mode of growth. These organisms present, in the exudation liquids, characteristic appearances to which we apply various terms, such as "colonies," "bacterium filaments," "dumb-bells," and "chains." The word *colony,* first introduced by Hallier, is used to designate groups of well-defined contour, in which the individual microzymes are held together by interstitial substance, which in this case forms a transparent matrix in which the rods and spheroids are embedded. A *bacterium filament* is simply a row of rods arranged end to end. A *dumb-bell* consists of two spheroids connected together by a bridge of envelope substance. A *chain* consists of a series of dumb-bells arranged end to end. Intermediate forms between dumb-bells and chains and rods or bacteria occur, which may be called *varicose rods.*

We have found it practically more convenient to use these common words † than to apply to the forms the specific desig-

* The paper was completed in June, 1872.

† Since the above was written, Professor Cohn, of Breslau, has proposed an improved classification and nomenclature of bacteria, which will probably be in future generally used by pathologists. See his 'Beiträge zur Biologie der Pflanzen,' pp. 127—224.

nations used by morphologists, *e.g.* *zooglæa* for colonies, *bacterium varicosum* for dumb-bells, *bacterium termo* for rods, &c.

As regards the morphological relation between these forms, little need be added to what has been stated in former papers. It is probable that they are organically continuous forms, but we are not in a position to state the precise relation in which they stand to each other.

It is, however, possible to divide them into two groups according to the circumstances under which they occur and thus to establish a classification, which, whatever may be its morphological significance, is of interest pathologically. Of these groups the rod is the type of one, the dumb-bell of another. In liquids in which the development of microzymes is going on with very great rapidity, as *e.g.* in the exudation liquid of intense infective peritonitis, single rods are found which are extremely minute, not larger than $\frac{1}{1000}$ millimeter in length. In exudation liquids (*e.g.* in the liquid of a subcutaneous abscess, or of a peritonitis which has lasted for some weeks) there are scarcely any rods; the prevalent forms are spheroids, dumb-bells, and chains.

The observations relating to exudation liquids have entirely confirmed the conclusion arrived at in my former report with reference to the microzymes of ordinary drinking water, that they do not either originate from fungi or develop to them. *On no single occasion has any form of fungus been discovered in any of the innumerable infective liquids, charged with microzymes which have been examined.*

PART II.—PREVIOUS EXPERIMENTS ON ACUTE INFECTION.

Before proceeding to bring under the notice of the Society the important results of the inquiries of the past year to which reference has just been made, I propose to give an account of some investigations made at former periods relating to the same subject.

In the year 1867 various experiments were made for the

purpose of comparing the effect of inoculating various pyæmic or septicæmic liquids with those obtained by the insertion of minimal quantities of chronically indurated lymphatic glands or other products of chronic infection. I transcribe the notes of some of these experiments.

August 23rd, 1867.—Three guinea-pigs and a dog were inoculated with purulent liquid from the ankle-joint of a patient affected with pyæmia. The patient, a man aged 28, was admitted into Middlesex Hospital under my care on the 20th of August, in a state of extreme typhoid depression. He complained of pains in all his joints and there was effusion in the left knee and ankle. The day after his admission he had swelling and inflammation at the root of the nose which subsequently extended to the eyelids. The inflamed parts were soon covered with phlyctenæ, which rapidly suppurated and a fœtid discharge from the nostrils commenced. During the next day subcutaneous suppurations presented themselves in various parts, particularly in the left arm and right leg. In these situations the skin was swollen and livid, while numerous phlyctenoid pustules appeared in the neighbourhood. Death occurred during the following night. At the post-mortem examination the pustular eruption was found to consist of separate pustules and of confluent groups, both of which were more abundant on the face, more especially around the nose than elsewhere. There was an abscess in relation with the middle third of the left clavicle which extended outwards for two inches behind and below the bone; no part of which, however, [illegible] ained to be in a rough or denuded [illegible] ous abscesses, of which a cons[illegible] were found to be lined [illegible] ffuse purulent infiltrat[illegible] dy. The serous [illegible] orrhagic patches [illegible] heart [illegible] a [illegible] surr[illegible]

by healthy pleura. Other similar subpleural nodules were met with in the middle and lower lobes, but in the latter instead of being grey throughout they were blood-stained externally, so that the outer part resembled on section an apoplectic mass. In the left lung the nodules were smaller and less numerous. The abdominal organs exhibited no morbid appearances excepting those of hyperæmia. The blood was examined microscopically and found not to contain an excess of colourless blood-corpuscles. In this case the pyæmic infection seemed to originate from the abscess near the left clavicle. The patient had complained of pain in this situation for ten days before any other symptoms presented themselves, during which time he was so free from constitutional ailment as to be able to attend to his occupation. He then began to suffer from pain in the joints, but had no serious symptoms until two days before his admission into the hospital. From the peculiar localisation of the cutaneous cellulitis and pustular eruption, and the fœtid coryza, I at once inferred that the case was one of acute glanders; but on careful inquiry found that the patient's mode of life was such as almost to preclude the possibility of this origin of the disease.

The first of the infected guinea-pigs died fifteen days after inoculation. The inoculation was performed in the usual way, care being taken to use a very small quantity of liquid. The animal was found on the 7th of September with hurried and difficult breathing and loss of power of the hind legs. It was at once killed. The dissection is noted as follows:—Wound: a large abscess, of very irregular form, exists at the seat of inoculation, measuring at its greatest diameter no less than two inches; it is lined by a well defined but thin membrane, and contains caseous pus. The scapular glands are not in the slightest degree enlarged and are perfectly transparent. The other subcutaneous glands are also healthy. Two of the mammæ are much swollen, and are found when cut into to be infiltrated with pus which is partly creamy, partly caseous. Lungs healthy. The liver contains hard nodules as large as pin heads, which are scattered over the

convex surface of the right and middle lobes. The spleen is much enlarged. Five or six nodules, varying in diameter from half a line to a line, project from its convex surface. Both the nodules in the liver and those of the spleen have the character of abscesses: they have firm capsules and contain pus which cannot be distinguished from that contained in those underneath the skin; they lie for the most part immediately underneath the serous membrane. The second guinea-pig died twenty days after inoculation.* A large abscess exists at the seat of inoculation; a similar abscess is found over the gluteal muscles of the right side, containing creamy pus, enclosed in an extremely thin capsule, the surrounding tissue being slightly hyperæmic, but not indurated. There is a third abscess between the intercostal muscles and the pleura, which projects into the pleural cavity. The axillary, tracheal and submaxillary glands are natural: the inguinal glands on the right side are slightly enlarged. On the surface of the lower lobe of the left lung there is a nodule of consolidation. On cutting into it it is found to be intensely hyperæmic, and to have the characters of pneumonic consolidation. A layer of false membrane adheres to its pleural surface. Besides this there are grains of much firmer consistence which project above the pleura. Of these, four can be counted on the lower and one on the upper lobe, while in the right lung a group present themselves near the outer margin of the lower lobe. One of them, larger than the rest, contains creamy liquid in its centre. On the convex surface of the liver nodules similar to those described in the other animal exist. The spleen is enlarged. —The dog, a small terrier weighing 8 lbs., died forty-nine days after inoculation, [illegible] of great exhaustion and emaciation. The inocu[illegible] be discovered, and the subcutaneous ly[illegible]lthy. The lesions of the internal [illegible] the exception that minute [illegible] its convex surface, the liver [illegible]

* Whenever the [illegible] is used. It is, h[illegible]

is much enlarged, and contains groups of pale grey nodules in considerable numbers. The organ is adherent to the anterior wall of the abdominal cavity, as well as to the omentum and diaphragm, by softish false membranes, and the whole organ is covered with a layer of soft "lymph."—From one of the guinea-pigs two others were inoculated, both of which died within forty-eight hours after inoculation. Pus taken from the seat of inoculation was used to inoculate two others; one lived a month the other forty-five days. In both there were multiple abscesses under the skin; in the one which lived longest the right lung exhibited nodules and patches of lobular condensation with pleural adhesions. In the other the internal viscera were healthy.

On the 15th of December, 1867, four guinea-pigs were inoculated with purulent liquid from the knee-joint of a man who had died of pyæmia. The patient, aged 42, was admitted into St. George's Hospital on the 20th of July with disease of the right astragalus and os calcis. Pyæmic symptoms supervened on the 3rd of December, and he died on the 14th. The liquid was taken and used about twenty hours after death. It was of a pale yellow colour, and opalescent appearance. Many of the pus corpuscles it contained were charged with fat granules. [Nothing was noted as to the presence of bacteria.] Of the four guinea-pigs, No. 1 died on the twenty-first day, the appearances being as follows:—An open wound exists at the seat of inoculation—a discharged abscess. Around the wound and extending from it towards the middle line are several abscesses, some of which are confluent. The scapular gland of the side corresponding to the wound is enlarged, and contains pus. The other subcutaneous glands and the internal organs are healthy. No. 2 died on the twenty-second day; it was inoculated in two places. Both wounds are open and have indurated borders. Numerous small subcutaneous abscesses are scattered over both flanks in the neighbourhood of the wounds. The right middle lobe of the left lung contains in its depth a hard nodule about two lines in diameter. There are one or two subserous nodules in the spleen. The other

organs are healthy. No. 3 died on the twenty-sixth day. The wound is open, and a cord of induration extends from it for three quarters of an inch to an abscess which is in immediate contact with the scapular gland, lying between it and the wound. The abscess measures five lines in diameter, and the gland, which is softened and contains creamy pus, is nearly as large. The other subcutaneous glands are healthy. [In this animal the internal organs were destroyed by its companions, so that they could not be examined.] No. 4 died on the twenty-seventh day. A very large abscess exists at the seat of inoculation, and a smaller one in its immediate neighbourhood. The scapular gland of the same side is enlarged and softened. The internal organs are found, on scrupulous examination, to be healthy.—On the 18th of November a guinea-pig was inoculated with pus from a pyæmic abscess in the posterior mediastinum of a patient who died in hospital on the previous day. The bodies of several dorsal vertebræ were diseased, and their excavated and roughened surfaces formed part of the wall of the abscess. There were pyæmic nodules in the lungs and kidneys. The animal was killed thirty-two days after inoculation. An abscess three or four lines in diameter, containing creamy pus enclosed in a membranous capsule, exists at the seat of inoculation. The capsule is firmly adherent to the skin and panniculus. The subcutaneous lymphatic glands are not enlarged, and the internal organs are healthy.

To these observations made in 1867-8, I append another of more recent date. A girl, aged 16, was admitted into the London Hospital on the 3rd July, 1870, with strumous disease of the bones of the little finger and great toe. On the 12th of January, 1871, the diseased organs were removed. The wound of the foot healed favorably, but that of the hand became unhealthy. Pyæmic symptoms (infective fever) appeared on the 26th. On the 7th of February an abscess was opened in the neighbourhood of the elbow-joint. Pus was collected in calcined glass tubes, which were immediately sealed. The same day the pus so collected was

injected into the peritoneal cavities of several guinea-pigs according to the following method:—In each case a sharpened canula previously boiled is first introduced through the anterior abdominal wall into the peritoneum. A capillary tube open at one end and expanded at the other into a closed bulb having been prepared, the pus is discharged into a calcined cup of glass, like that used for holding vaccine in my former experiments. The open end of the capillary tube is then dipped into the pus, and filled by capillarity to a certain distance. [If desired the quantity taken up can be easily determined by the balance.] The charged end is then passed by the canula into the peritoneum, and the bulb slightly warmed, by which means the whole of the pus is discharged.—Nine weeks after inoculation two of these animals were dissected. Both exhibited the same appearances. The lesions observed (in No. 1) were as follows:—At the seat of puncture there is a slight induration. The inguinal glands are enlarged and softened on both sides; the scapular glands are also somewhat enlarged; but the other subcutaneous glands are normal. The inguinal glands, particularly on the right side, are surrounded by zones of intense injection, and there are numerous minute abscesses disseminated under the integument in their neighbourhood. Another group of abscesses surrounds the internal inguinal ring. The peritoneum is distended with sero-sanguineous liquid, in which are seen active bacteria of small size but very distinct form. The omentum is beset with minute nodules. The centrum tendineum of the diaphragm also exhibits a few nodules. The liver weighs thirty-five grammes: it is very pale and beset here and there with nodules, some of which are purulent. The spleen is enormously enlarged. [Condition of mesenteric glands not noted.] The pleuræ and the pericardium contain exudation liquid. Groups of opaque grey nodules are scattered throughout the lungs, both in the depth and at the surface.

The observations related in the preceding paragraphs show that although, as already stated, there is a close correspondence as regards the distribution of the infective nodu

the internal viscera between animals infected with pyæmic products and those inoculated with equally small quantities of the material derived from chronic secondary indurations, the contrast between the results is very marked. I will refer first to the lesions, and secondly to the development and mode of termination of the two processes.

Subcutaneous lesions.—When an extremely small quantity of chronically enlarged lymphatic gland is inserted under the skin in the manner described in my former paper, no abscess is produced either at the seat of inoculation or in any other situation. The wound to all appearance heals; but if it is carefully examined many weeks after, it is found, provided that the ingrafting has been successful, that a group of semi-transparent nodules exists under the skin, which represent the centre of infection. In the pyæmic cases there may also be induration of the same nature, but the nodules assume from the first the character of abscesses, and are accompanied by groups of smaller abscesses, disseminated in the neighbouring tissue. In a considerable proportion of the pyæmic animals these primary and secondary metastatic subcutaneous abscesses were the only lesions found, even though the dissections were made many weeks after inoculation.

Visceral lesions.—In chronically infected animals (using the term for animals infected by the insertion of minimal quantities of fresh induration material in the manner set down in the preceding paragraph) the visceral changes are much more uniform and characteristic. To judge of the difference between them and the corresponding pyæmic le[illegible] [illegible]ral organs must be separately referred to.
[illegible] liver (*see* 'Eleventh Report of
[illegible]uncil,' p. 110) does not
[illegible] which the term
[illegible]larges uniformly
[illegible] interstitial
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Even when the change does not extend to the whole organ, but affects only a certain number of lobes, the newly-formed tissue retains what descriptive pathological anatomists call the infiltrated as distinguished from the disseminated arrangement. In the acute affection the liver lesion is characterised by the presence of disseminated nodules, firm externally, purulent internally. (2.) The difference between the tuberculous lung and the pyæmic lung is perhaps not so obvious, but the characteristic appearance of the semi-transparent "iron grey nodules" (*loc. cit.*, p. 114), their uniform distribution throughout the parenchyma of the lungs, and the absence of concomitant pleuritic exudation or false membranes are facts which render the contrast between the two conditions sufficiently decided. (3.) Another very important distinction may be based on the condition of the lymphatic glands: in chronic (tuberculous) infection the internal lymphatic glands always become diseased tertiarily. They enlarge and pass into the state of chronic fibrous induration (*see loc. cit.*, p. 103), eventually becoming opaque and caseous. These changes may be studied in the bronchial glands, but particularly in those which receive their tributaries from the liver, in respect of both of which it may be observed that during the first two or three months after inoculation the glands remain unaffected. It is not until the pulmonary or hepatic disease has made some progress that the corresponding changes in the glands begin. In the acute affection it is entirely different. All the lymphatic glands which are in relation with the inflamed serous membranes become the seat of acute enlargement and softening. In tuberculosis they do not undergo this change, because the serous membranes are not inflamed.

If we turn our attention from the lesions to the development and mode of termination of the two morbid processes, the contrast appears to be even greater. The most striking fact in this relation has been already referred to, viz., that when minimum quantities of induration material are used, the graft apparently lies hidden where it was inserted for many weeks, producing neither local inflammation nor fever

(*loc. cit.*, p. 105). It is not till weeks have passed away that it begins to show its effect, first, by the enlargement of the lymphatic glands in relation with the centre of infection, and then by the slowly progressing changes above referred to in the internal organs. The fatal result may, as my former observations showed, be postponed to an indefinite period, the animal living on in apparent health so long as there are sufficient of its organs unspoilt by the disease for the continuance of their functions. When it dies the result is usually determined by the setting up of an acute infective process, manifesting itself in serous inflammations, in the rapid softening of the indurated parts, and in the formation of vomicæ or abscess-like nodules in the diseased organs. Consequently, if we wish to study the chronic alterations of the internal viscera as they are, we must not wait till the animal dies, for by doing so we are almost sure to find them materially modified by the acute inflammatory softenings which precede the fatal issue. Just as in human pulmonary tuberculosis, so in the artificial disease, death is not caused by chronic induration, but by the acute infective inflammations which supervene upon them, or to which they furnish the occasion.

Part III.—Experiments made in 1872 by Dr. E. Klein.

It has been already stated that in an experiment made in the spring of 1871 it was found that in animals infected by the introduction of a minimal quantity of pyæmic pus into [illegible] with [illegible] possible precautions against conta- [illegible] contact with other media, [illegible] found to be charged with [illegible] important question. [illegible] a characteristic [illegible] of the peri- [illegible] were made [illegible] ted in [illegible] uda-

tion liquids but of chemical excitants, particularly dilute ammonia and concentrated solution of iodine in hydriodic acid. As regards the ammonia, precautions were taken to guard against contamination by boiling and cooling the liquids as well as the implements to be used immediately before injection. In the case of the iodine solution this was of course unnecessary. In every instance it was found that the exudation liquids, collected from twenty-four to forty-eight hours after injection, were charged with bacteria; whence it appeared probable that the existence of these organisms was dependent, not on the nature of the exciting liquid by which the inflammation was induced, but on the intensity of the inflammation itself.

After an interval of several months, occupied in other investigations, the inquiry was resumed. The experiments made may be divided into two series; in one those are comprised in which the liquids of acute infective inflammations were used, and consequently the results exhibited those characters of intensity and virulence which are expressed by ~~the term septic~~æmia; in the others the results resembled those which have been already referred to in the preceding section both as regards the methods and the phenomena induced. The important conclusions to which they have led will be stated after the facts themselves have been discussed. The following is a tabular summary of the experimental results of the first series:—

TABULAR SUMMARY *of* TWENTY-SEVEN OBSERVATIONS *relating to the action of the exudation-liquids of acute infective inflammations when introduced into the peritoneum or into the venous system.*

Number.	Case.	Description of exudation-liquid used.	Designation of test animal.	Date of dissection.	Duration of induced inflammation.	Mode or channel of introduction of infecting liquid.	Quantity of infecting liquid used. +	Result.
1	Jan. 9	Liquid obtained by puncture from the cavity of the uterus of a bitch affected with acute infective muco-enteritis and metritis. The animal was killed moribund, and immediately afterwards dissected. The liquid was charged with rods and dumb-bells.	Guinea-pig, 11	Jan. 10	c. 20 hours	Peritoneum	5 Div. (c. 6 ♏)	Intense peritonitis; serous surfaces of stomach and intestines hyperæmic; sanguinolent exudation-liquid in peritoneal cavity. Exudation-liquid crowded with rods, along with a certain number of dumb-bells, chains, and colonies. Blood plasma particulate, the particles in tremulous movement.
2	Feb. 26	Purulent liquid from diffuse subcutaneous suppuration in groin of a rabbit, in which blood had been taken from the crural artery ten days before. The *plasma puris* is charged with rods and colonies of dumb-bells; the blood contains numerous minute rods, and here and there a dumb-bell. The animal was killed moribund, and immediately dissected.	Guinea-pig, 28 *a*	Feb. 27	12 hours	Ditto	3½ Div. (4 ♏)	Intense peritonitis; exudation-liquid milky, crowded with minute rods.

Number.	Case.	Description of exudation-liquid used.	Designation of test animal.	Date of dissection.	Duration of induced inflammation.	Mode or channel of introduction of infecting liquid.	Quantity of infecting liquid used. +	Result.
3	Feb.26	Purulent liquid from diffuse subcutaneous suppuration in groin of a rabbit, &c.	Guinea-pig, 28 *b*	Mar.12	16 days	Peritoneum	3¼ Div. (4 ♏)	Intense peritonitis, with false membranes on surfaces of viscera, and extensive adhesions; intense pleuritis; infective nodules (abscesses) in the abdominal viscera and underneath the peritoneum. Serous exudation-liquids abundant, crowded with rods, and containing other forms in small numbers; that of the pleura exhibits the particulate character. The purulent liquid of the pyæmic nodules in the liver contains rods in number, along with dumb-bells and chains. Blood tenacious, it contains numerous minute rods.
4 and 5	„ 28	Peritoneal milky exudation-liquid of 28*a*.	Guinea-pig, 30 *a* & *b*	Feb. 28	10 hours	Jugular vein	5 Div. (6 ♏)	Both animals became collapsed soon after injection. Intense peritonitis; exudation-liquid crowded with rods of various sizes.
6	Mar.12	Pleural exudation-liquid of 28 *b*.	Guinea-pig, 39 *a*	Mar.26	14 days	Peritoneum	2½ Div. (3 ♏)	Intense peritonitis and pleuritis, with false membranes covering surfaces of thoracic and abdominal viscera; consolidation of both lungs; pericarditis; pleural and peritoneal exudation-liquids crowded with bacteria.
7	„ 26	Pleural exudation-liquid of 39 *a*.	Dog, I.	...	...	Ditto	5 Div. (8 ♏)	This animal, after exhibiting for several hours the phenomena of acute infection (*i. e.* rigors followed by collapse, with vomiting and purging), rapidly recovered.
8	„ 20	Purulent liquid from diffuse subcutaneous suppuration produced by	Guinea-pig, 49 *a* & *b*	Mar.20	6 hours	Ditto	3 Div. (3½ ♏)	Both animals became collapsed shortly after injection. Intense peritonitis; clear yellow viscid exudation-liquid.

			injection of $3\frac{1}{2}$ ♏ of dilute liquor ammoniæ under the skin of a guinea-pig. The liquid was charged with minute bacteria.							crowded with minute rods.
9	„	20	Ditto.	Guinea-pig, 49 *c*	„	21	24 hours	Ditto	Ditto	Post-mortem appearances the same.
10 and 11	„	21	Peritoneal exudation-liquid of 49 *c*.	Guinea-pig, 50 *a* & *b*	„	22	ditto	Ditto	Ditto	Intense peritonitis; exudation-liquid viscid, and crowded with minute bacteria.
12	„	23	Peritoneal exudation-liquid of 50 *a*.	Dog, XV *d*	„	23	6 hours	Ditto	15 Div. (18 ♏)	Death preceded by collapse, cramps, vomiting, and diarrhœa. Peritonitis and hæmorrhagic gastro-enteritis.
13 and 14	„	16	Purulent liquid from the enlarged and softened mesenteric glands of 42 *a*.	Guinea-pig, 44 *a* & *b*	„	18	2 days	Ditto	4 Div. (5 ♏)	Intense peritonitis; purulent false membranes on the serous surfaces of the intestines and abdominal viscera; exudation-liquid viscid, swarming with minute short rods. Mesenteric glands enlarged and softened; one of them contains a large abscess, the purulent contents of which are charged with bacteria.
15	„	18	Purulent liquid from the enlarged and softened mesenteric glands of 44 *a*.	Guinea-pig, 46 *a*	„	19	24 hours	Ditto	$4\frac{1}{2}$ Div. ($5\frac{1}{2}$ ♏)	Intense peritonitis; exudation-liquid crowded with minute rods, other forms distinguishable with difficulty.
„	„	18	Ditto.	Guinea-pig, 46 *b*	„	29	11 days	Ditto	Ditto	Intense peritonitis with adhesions; infective nodules in omentum, mesenteric glands, and viscera; exudation-liquid viscid and crowded with rods, which also exist in number in the purulent contents of the nodules.
16, 17, & 18	„	19	Peritoneal exudation-liquid of 46 *a*.	Guinea-pig, 47 *a*, *b*, & *c*	„	20	12 hours	Ditto	5 Div. (6 ♏)	All three animals became collapsed shortly after injection. General peritonitis; serous surfaces of intestines intensely injected; viscid exudation-liquid, crowded with rods.

Number.	Case.	Description of exudation-liquid used.	Designation of test animal.	Date of dissection.	Duration of induced inflammation.	Mode or channel of introduction of infecting liquid.	Quantity of infecting liquid used. +	Result.
19	Mar. 19	Peritoneal exudation-liquid of 46 *a*	Dog, VI *d*	Mar. 19	4 hours	Peritoneum	10 Div. (12 m)	The same symptoms during life and appearances after death as in No. 12 (see commentary).
20	„ 20	The same liquid kept since the preceding day in hermetically closed tubes.	Dog, VII *d*	„ 20	$4\frac{1}{2}$ hours	Ditto	Ditto	The same.
21	„ 20	Ditto.	Cat, VIII *e*	„ 21	5 hours	Ditto	Ditto	The same.
22	„ 20	Ditto.	Cat, IX *e*	„ 21	About 12 hours	Ditto	Ditto	The same.
23	„ 21	Ditto.	Dog, X *d*	„ 21	About $6\frac{1}{2}$ hours	Ditto	6 Div. (7 m)	The same.
24	„ 21	Ditto.	Dog, XI *d*	„ 22	About 12 hours	Jugular vein	5 Div. (6 m)	The same. Symptoms extremely marked, and morbid changes excessive.
25	„ 21	The same liquid diluted with $\frac{3}{4}$ per cent. solution of chloride of sodium.	Cat, XII *e*	„ 22	ditto	Peritoneum	20 Div. (24 m)	The same.
26	„ 21	Ditto.	Cat, XIII *c*	„ 24	About 3 days	Jugular vein	10 Div. (12 m)	The same.
27	„ 22	Blood of XI *d* charged with bacteria.	Dog, XIV *a*	„ 22	19 hours	Peritoneum	15 m iv. (18 m)	The appearances on dissection indicate a peritonitis of extreme intensity. Surface of stomach and intestines covered with purulent false membranes; omentum intensely injected, and in parts in a state approaching gangrene; intense muco-enteritis of whole intestinal tract. Pleuritis, with sanguinolent exudation-liquid. Blood viscid, containing numerous minute bacteria.

Explanation of the Table.

In the first eight vertical columns the circumstances relating to the introduction of the exciting liquid are stated; in the last column the result as regards the conditions of the serous membranes and internal organs, and of the blood. The liquids used as excitants were in each case exudation liquids; they were injected immediately after removal from the living animal. Of the twenty-seven experiments, serous liquids were employed in eighteen; liquids of subcutaneous suppurations in four; liquids from softened infective nodules and lymphatic glands in three. In one, blood of the infected animal was used; and in another the contents of an infectively inflamed uterus. All of these liquids were charged with bacteria at the moment that they were removed from the living animal. In the fourth column the test animals, or re-agents, are designated by numbers; the Arabic numerals relate to guinea-pigs, the Roman to cats or dogs. The fifth column gives the time after inoculation at which dissection was performed. In most cases the test animals were killed for the purpose when *in articulo*, in order that the blood and exudation-liquids might be examined in the recent state. In a few cases the dissections were made immediately after the animals had died naturally. From the sixth column it appears that in four cases the infecting liquid was injected into the jugular vein; in the rest into the peritoneum. The quantities used varied from 3 minims to 24 minims (consequently they were always incomparably greater than those employed in experiments of 1867-68, which never exceeded a fifth of a minim).

Although the liquids were similar, all being products of rapidly progressing infective inflammations, the induced results were not all of equal intensity. Of the twenty-seven animals used as re-agents, twenty died or became moribund [illegible] hours, the mean duration of life after [illegible]t thirteen hours. The others lived

various periods from two days to sixteen days. The protraction of the fatal result in these instances cannot be attributed to anything special in the particular experiments, for the table shows that in other animals injected at the same time and with the same material, no such postponement was observable. The facts illustrate a general principle which we shall see otherwise exemplified—that if an animal survives the first outburst of infective fever, it may be many days before it is exhausted by the subsequent secondary inflammations.

Of the twenty animals which died within twenty-four hours after infection, all, excepting three (two guinea-pigs and a dog), received the excitant in the peritoneal cavity. In these three, in which the exudation-liquid was injected by the jugular vein, the signs of serous inflammation were quite as decided as in the others. All the twenty-seven animals had intense peritonitis, and in the two guinea-pigs injected by the jugular vein, both of which were moribund before the end of the day, it was just as intense as in any of the others. In the dog, in which the process was equally precipitate, it was specially noted that the changes observed on dissection happened to be more pronounced than in the animals of the same species which had received the same liquid directly into the abdominal cavity. Hence, although the local action of the exciting liquid cannot be regarded as insignificant, it is not the determining cause of the intensity of the peritonitis. This conclusion is confirmed by the observation that the pleura and pericardium were often as intensely affected by injections into the abdominal cavity as the peritoneum itself.

From the fact that in all rapid cases of acute infection there is intense peritonitis, it might be surmised that the peritonitis is the immediate cause of death. Such an inference would be groundless and probably erroneous. The symptoms which we recognise in the dog as those of intense infective fever, viz. collapse attended with vomiting and purging, manifested themselves so early in all the cases, and particularly in the animals which were injected by the

jugular vein, that it would be difficult to regard them as secondary to the peritonitis. The rapidity and completeness of the recovery which takes place in some instances (*e. g.* in dog No. I) after collapse of some duration, affords additional ground for attributing the collapse itself, with its accompanying phenomena, to the direct action of the infective agent.

In all the very rapid cases the peritoneal liquid exhibited the same character. It was viscid, and coagulated imperfectly or not at all. It contained pus corpuscles, but they were relatively not numerous; the plasma (*liquor puris*) often exhibited a tremulous movement which, under high powers, is found to be due to the presence of minute rods. With lower powers particles cannot be distinguished; all that can be seen is the peculiar tremulous movement of the liquid, which, however, is sufficiently characteristic. In addition to this appearance, the liquid always contains other actively moving rod-like bodies, larger than the particles above mentioned, but still not greater than $\frac{1}{500}$ of a millimeter in length. In the most rapid cases dumb-bells and chains are mostly absent, but they soon appear if the peritonitis lasts long enough, or if the liquid is kept in the warm chamber in a plugged eprouvette. In all the acutely infected animals to which the table relates in which the blood was examined, it exhibited microscopical appearances which were characteristic and unequivocal. They were of two kinds, and corresponded to those already referred to as presenting themselves in the serous exudation-liquids. The blood plasma exhibited more or less distinctly the tremulous movement of particles above described, and contained short rods. It was also observed that colourless corpuscles often contained rods in their substance and that the blood discs seemed to adhere to each other, the blood possessing a remarkable viscidity, the nature and significance of which will be a subject for future investigation.

The symptoms observed during life, and the changes found on dissection in acute phlegmonous infection, which terminate in less than twenty-four hours, differ considerably

according to the animal used. In guinea-pigs collapse comes on very rapidly after injection, whether into the venous system or into the peritoneum. It is marked chiefly by loss of muscular power and diminution of temperature. After death there is intense peritonitis. In the dog or cat the phenomena are more marked. In some few cases the animal passes rapidly into the state of collapse, but more commonly an hour or two elapses before any very striking effect is observable. Among the earliest obvious phenomena are muscular twitchings and shiverings, which may come on during the second or third hour; but thermometrical measurements in the rectum show that the temperature rises from the first. The supervention of collapse is indicated by failure of muscular power. This is accompanied by retching and vomiting, which are soon followed by diarrhœa. The retching is associated with violent spasmodic muscular movements. As soon as the contents of the stomach have been expelled, a frothy greenish liquid of viscid consistence begins to be discharged. The diarrhœa is at first attended with tenesmus, but afterwards becomes colliquative. The alvine discharge is mucous, shreddy, and always more or less stained with altered blood. A few hours before death the temperature begins to sink, eventually falling below the normal; this depression begins soon after the accession of the more severe symptoms, so that the period at which the vomiting and purging come on with violence usually corresponds to the acme of temperature. The most important of the changes observed after death are those which correspond with the symptoms. There is intense muco-enteritis of certain sections of the intestinal tract, the parts chiefly affected being the duodenum and rectum. The cases in which the alterations are most marked, are those in which, perhaps from great endurance on the part of the subject used, the toxic action, although equally intense, has been resisted a greater number of hours, so that it has had more time to develop itself. In such cases the whole tract may be inflamed, but in general the changes are limited as above stated. The mucous membrane

of the duodenum is uniformly and intensely reddened, and its cavity distended with a mucous, more or less blood-stained, frothy liquid, in which float large flakes of exfoliated epithelium. The state of the rectum is similar, the staining of the mucous membrane is as deep and uniform, but the liquid is of a darker colour. The jejunum partakes more or less of the same conditions; but the ileum is always freer from change than any other part. Peyer's follicles are sometimes enlarged. The peritoneum is always hyperæmic in the most severe cases, and particularly the protracted ones, the hyperæmia is intense, and there are hæmorrhagic spots particularly on the omentum and mesentery. The characters of the exudation-liquid have been already referred to.

These symptoms and appearances closely correspond, if they are not absolutely identical, with those of septic infection in the dog, *i. e.* with those which are produced by the injection of putrid animal liquids into the venous system. To illustrate this, I take the following account of the post-mortem appearances in this kind of poisoning from the well-known research of Bergmann on this subject.* After the injection of four to seven cubic centimeters (60 to 100 minims) of putrid blood carefully filtered, into the venous system of a large dog, the animal dies in from four to ten hours, the fatal issue being preceded by collapse, vomiting, and diarrhœa. On dissection, says Bergmann (p. 14), "the most important post-mortem appearances are those found in the intestine," consisting in "intense reddening and swelling of the mucous membrane." In general the alteration "does not extend to the whole intestine, but affects the pyloric end of the stomach, the duodenum, the upper coils of the jejunum." . . . "The intestine is filled with reddish thick mucus, which strikingly resembles the rose-coloured rice water of the cholera stool. The further down in the intestinal tract the liquid is examined the darker is its colour, until in the large intestine it assumes a dull brownish-red tinge which is identical with that of the last alvine

* Dr. E. Bergmann, 'Das putride Gift und die putride Intoxication.' Dorpat, 1868.

dejections. The quantity of transudation-liquid in the intestine is very considerable. Even when the extent of morbid alteration is greatest, it is mostly confined to the upper part of the small intestine, colon, and rectum; the middle part of the small intestine is either quite free or very slightly affected. . . . The greater the intensity and extent of this genuine gastro-enteritis, the larger is the quantity in which shreds of separated epithelium are mixed with the intestinal contents, and the more abundant the excoriations and exfoliations of the mucous surface." . . . "In the worst cases an enteritis of equal intensity extends the whole way from the cardia to the anal orifice. The croupous diphtheric affection asserted by some observers to exist, I have not observed. It appears to me that the yellowish shreds which separate from the mucous surface are merely bits of shred epithelium."

The agreement of this description so far as relates to the mucous membrane of this intestine is complete. There is, however, one point of difference. Bergmann did not as a rule observe peritonitis in his animals. He records, indeed, that the mesenteric vessels were distended with blood, and that there were "sugillations" both in the mesentery and omentum; but it was only in two instances that he found the peritoneum generally inflamed. In these animals the exudation is described as being of a dirty brown colour, and very abundant; from which it seems reasonable to infer that in many other cases the condition was not observed, for it is scarcely probable that if the peritoneum was, as a rule, normal, it would be found in these two instances so intensely affected. It is much easier to believe that the very inconsiderable exudations which usually present themselves were overlooked.

In all other respects the differences are too inconsiderable to require special notice.

The inferences above derived from the comparison of the phenomena of septic infection in the dog with those of acute phlegmonous infection, as to their close relation with each other is confirmed by the following experiment, in which

guinea-pigs were injected with exudation liquids which had been kept for six days at the temperature of the body. It is seen that the results produced by such a liquid in the putrescent state, are not distinguishable from those which it produces when injected fresh in the same manner.

14th March.—Purulent liquid obtained from the spleen of a guinea-pig, which had been diluted with three quarters per cent. solution of common salt, and kept at a temperature of 40° C. since the 8th, was injected into the peritoneal cavities of two other animals of the same species, the quantity used in each case being six minims. Both were moribund on the 16th. In the first there was intense peritonitis, and the peritoneum contained the usual viscid exudation-liquid. In addition to this there was œdema of the whole anterior half of the trunk. The blood-vessels were dilated and distended with stagnant blood, over a region corresponding to that of the œdema, and extending from the middle line towards either flank. Beyond this region the vessels were remarkably empty. Not only the peritoneal liquid, but that of the subcutaneous œdema, was crowded with bacteria. The same forms were to be found in numbers in the blood, the plasma of which exhibited the particulate appearance already so often referred to. In the second animal, with the exception of the subcutaneous œdema, the appearances were similar. The blood and exudation-liquids exhibited the same characters.

I have now to refer to the second series of observations in which the infecting liquids were not products of virulent inflammation, such as those described in the last paragraph, but of more slowly progressing inflammatory processes, chiefly characterised by softening and unhealthy suppuration, either of infective nodules or of previously consolidated or infiltrated tissues. In the three series of experiments, which will be given as examples of the rest, the original source of the infecting material was the diseased lungs of human beings or animals affected with chronic pulmonary tuberculosis in the stage of softening. The animals thus infected

served in their turn as sources for the infection of others, the liquids used being derived from softened pyæmic nodules or lymphatic glands.

December 22nd.—A dog died in the Hospital for Animals of pulmonary phthisis of long duration. The condition of the left lung and pleura is noted as follows:

There is a patch of consolidation about half an inch in diameter, over which the pleura is firmly adherent. Other similar nodules exist in other parts of the surface of the organ. On making sections it is found that each of these nodules is softened at its centre, and that around each, the lung substance is infiltrated as well as disseminated "with grey and yellow tubercles." Of the remainder of the organ, the greater part is airless. The pleural cavity contains several ounces of clear liquid. Of the pleural exudation-liquid a few minims were injected into the peritoneal cavity of each of five guinea-pigs. Three others received a similar quantity of the purulent liquid from one of the softened nodules. The liquids were injected undiluted by means of a Pravaz syringe, the quantities used being relatively large. The animals were dissected respectively, fifteen, twenty-three, twenty-eight, thirty-four, fifty-nine, sixty-six, sixty-nine, and sixty-nine days after injection. The lesions were substantially the same in all, in every instance the serous membranes were inflamed and contained variable quantities of exudation-liquid charged with bacteria, in most cases there were extensive adhesions and false membranes; all of the organs contained infection nodules, which possessed the characters already described, being hard and firm externally with soft and purulent centres. The pus contained in them was always charged with microzymes both in the form of rods and in those of dumb-bells and chains. The latter forms prevailed most in those animals which had survived longest. In those liquids in which they were either absent or in relatively small numbers when the liquid was fresh, they multiplied rapidly when the liquid was placed for twenty-four hours in a warm chamber at the temperature of the body, while the rods seemed to undergo a corresponding

diminution in numbers. This fact was observed very frequently in infective liquids so cultivated.

The contrast between the conditions observed in these experiments and those which characterise the chronic infection which is produced by the injection of minimal quantities of induration material is very striking. Here all of the lesions are the products of an acute process. This may be inferred from their characters, but we have a more positive ground for the conclusion that they are so in the fact that, in the animal which died at the end of a fortnight after injection, the infective abscesses in the internal organs were already fully developed, and that as regards the state of the serous cavities the appearances did not differ in any material respect from those which were observed in animals which survived forty, fifty, or even sixty days. Clearly, if in the former case the mischief was done rapidly, we must believe that it was also done rapidly in the others, and that if we had had the opportunities of examining those animals which lived longest at the same early stage in the disease we should have found similar lesions. In either case we have to do with the products of a rapidly developed pyæmia, the lesions of which not only come into existence but attain their full development during the first few weeks after injection, the difference between the long cases and the others amounting to nothing more than that in the former the struggle was more protracted. Essentially the processes are the same. If one is acute the other is.

The points of distinction referred to in the introductory section between acute and chronic infection (pyæmia and tuberculosis) are also well illustrated in the experiments now under consideration. Two perfectly conclusive facts may be referred to in evidence that the process with which we are now concerned is not that which I described in my former papers. The one is that the lesions of the internal organs although they accord with each other with the greatest exactitude as observed in the different animals of the same series, notwithstanding the differences of duration of the morbid processes, are entirely different from those described

by me as characteristic of "artificial tuberculosis." The second is that, in the experiments now before us, the animals were the subjects of serous and other inflammations from the beginning, whereas in tuberculosis no lesion whatever is discoverable by microscopic examination until the fourth or fifth week after infection.

Dec. 30th.—Material derived from the lung of a patient who had died the day before of phthisis in the third stage, in the Hospital for Consumption, was injected into the peritoneum in three guinea-pigs, and under the skin in three others. The first three were dissected respectively, twenty-nine and forty-four days after inoculation, having either died of themselves or having been killed *in articulo*. The other two were killed, fifty-four and sixty-eight days after injection. In the first three the lesions corresponded very closely with those already described. The pleura and peritoneum were inflamed and contained considerable quantities of exudation liquid. The liver, spleen, and lungs contained pyæmic nodules, and there were similar nodules in the mesentery, omentum, and centrum tendineum of the diaphragm. One of the abscesses in the omentum was of very large size. The purulent liquid from the nodules contained rods and dumb-bells in great numbers; the latter were found as usual to have become much more numerous after the liquid had been kept for a day in the warm chamber. The same forms were seen in the pleural and peritoneal liquids. In all of these animals the blood was found to present the usual alteratious. The plasma was particulate, the particles exhibiting the movement before described, and contained rods and dumb-bells. The other three experiments are of interest as showing how little the mode of insertion modifies the result as regards the internal lesions. They lived longer, but in other respects the morbid process was the same, and it is specially to be noted that the signs of pleural and peritoneal inflammations were quite as marked as in the others. Purulent liquid from one of the animals was injected in the usual way into the peritoneum of a healthy guinea-pig, which, on dissection three weeks

later, had peritonitis and infective nodules in the usual situations.

Feb. 27th.—A small female monkey died in the Hospital for Animals of pulmonary phthisis. The principal lesions were as follows:—In the thorax the morbid appearances are confined to the right side. The right pleura is everywhere adherent. The whole of the right lung, with the exception of the apex is either consolidated and airless, or beset with numerous disseminated nodules. These nodules are for the most part firm externally, but softened at their centres. Here and there, there are vomicæ. The peritoneum contains clear liquid, along the border of the colon there are several nodules varying in size from that of a pea to that of a hazel-nut. Of these the smaller are grey, the larger yellowish white on section. Similar but smaller nodules exist in the mesentery. In the liver there are numerous softened nodules as well as round the portal vessels. Purulent liquid from the pulmonary nodules containing microzymes of both forms was injected in the usual quantity into the peritoneal cavities of five guinea-pigs. The tests were dissected at periods varying from thirty to forty-three days after infection. The post-mortem appearances, both as regards the serous membranes and the internal viscera, resembled in all respects those already described.

Conclusion.

Considering the complexity of the subject we have attempted to investigate, and the importance of the questions involved in it, it is requisite to exercise extreme caution in drawing conclusions. There are, however, one or two propositions which may be stated with confidence as the results of our observations. It has been shown (1) that that combination of malignant fever with intense and destructive inflammation to which pathologists have rightly applied the term septicæmia, because it is known by experiment as well

as by clinical observation to result from the existence in the blood of putrescent albuminous matter, may also be produced by the introduction, into the circulation or into the serous cavities, of small quantities of liquids derived directly from living tissues in certain states of inflammation; and that such states have the same distinctive characters as those which distinguish inflammation of septicæmic origin. (2.) That pyæmia (the term being understood to denote a general febrile disorder of less virulence than that of septicæmia, accompanied by numerous disseminated inflammations, characterised chiefly by their proneness to suppuration) is so closely related to septicæmia as regards its origin and essential nature that in these respects no line of distinction can be drawn between them; and that pyæmia, like septicæmia, may originate from a purely traumatic inflammation, independently of any infection with contagium derived from a previously existing pyæmic inflammation. (3.) That both of these conditions are characterised by the existence of microzymes in the infected liquids; and that the relation of intensity between different cases of septicæmia and pyæmic infection is indicated by the number and character of these organisms; so that in the most intense processes (*i. e.* those which exhibit the characters of septicæmia), the exudation liquids and the blood are crowded with actively moving bacteria, while in the more chronic processes, the spheroidal and dumb-bell forms prevail, and the numbers of organisms found in the liquids are relatively inconsiderable.

Among the more important of the further subjects of inquiry which our researches suggest, are the relations between pyæmia and other states of disease, and particularly ordinary inflammation, and the relation of pyæmic or infective fever to the local inflammations with which it is associated.

The fact that ordinary traumatic inflammation may pass by an apparently gradual transition into a pyæmic or septic one, is evidence that the distinction is not always obvious, but does not prove that it is ill-defined or vague. For, although it may be extremely difficult to say precisely when

infective characters begin to manifest themselves, yet, if we are entitled to assume that the appearance of those characters is an evidence that an infective substance or contagium which was not there before, has come into existence in the blood or tissues, there must have been a moment at which that substance was introduced, and consequently a moment at which the process would have to be regarded no longer as the effect of the injury, but as the combined effect of the injury and the infection. In this way it is conceivable that the line between healthy and unhealthy inflammation may be in reality sharply defined, however faint and difficult to appreciate.

In our experiments of the year before last it was proved that, as a rule, the normal liquids of the animal body (blood, tissue, juice, &c.) do not contain microzymes either in germ or visible form. It was also proved that in common drinking water and in other watery liquids with which the body is constantly in contact, microzymes, although not distinguishable by the microscope, exist potentially, that is, in germ. From these facts, taken in combination with the existence of similar organisms in infective exudation liquids, the inference is very obvious that, inasmuch as these organisms cannot have originated from the normal tissues or juices, they must have been derived from the external moisture.

This inference would of course at once fall to the ground if it could be shown either that microzymes spring up of themselves, or that the forms which occur in the animal liquids are not specifically identical and organically continuous with those which exist in aqueous media outside. In the absence of any such proof, I am not aware of any objection to the assumption of their external origin and am quite willing to admit it, provided that it is clearly understood that, even if the extrinsic origin of microzymes were proved, it would afford no ground for concluding that the origin of the contagium itself is also extrinsic. It does not at all follow because these organisms come in from outside that they bring contagium along with them; for it may be readily admitted that they may serve as carriers of infection

from diseased to healthy parts, or from diseased to healthy individuals, and yet be utterly devoid of any power of themselves originating the contagium they convey.

CASE

OF

ABDOMINAL ANEURISM

SUCCESSFULLY TREATED BY PROXIMAL PRESSURE OF THE AORTA.

BY

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In the year 1864 Dr. William Murray, of Newcastle-on-Tyne, communicated to the Royal Medical and Chirurgical Society the particulars of a case of abdominal aneurism, cured by proximal pressure upon the abdominal aorta. The paper was read, and the patient exhibited, at a meeting of the Society held about six weeks after the operation. In common with many other Fellows of the Society, I examined the patient with great interest and felt the hard pulseless tumour which occupied the site of the previous aneurism. The patient remained well for six years, when by reason of failing to obtain any other employment, he resumed his former laborious occupation as a paviour, and in a very short time began to suffer from violent pain in the epigastrium, which was speedily followed by other symptoms of aneurism in that region.* This second aneurism was, however, found to be situated so close to the diaphragm that it was impossible to compress the aorta between it and

* 'The Rapid Cure of Aneurism by Pressure.' By W. Murray, M.D., &c. London. Page 24.

the heart, and the patient died suddenly in the course of the same year.

Although the details of the post-mortem examination were shortly afterwards published by Dr. Murray, yet, as they are not recorded in the 'Transactions' of the Society, it may be desirable briefly to recapitulate them here. The original aneurism was found to have been situated in that portion of the abdominal aorta from which the inferior mesenteric artery is given off, and its remains consisted merely of a fibrous mass. The trunk of the inferior mesenteric artery had dwindled to the size of the radial artery and its coats were thin and flaccid; the circulation through its branches having been carried on by means of the connection between the colica sinistra and colica media arteries, both of which were enormously enlarged. Complete collateral circulation had been effected by the enlargement of vessels both outside and inside the abdominal cavity. The enlarged vessels on the front and sides of the abdomen had been seen pulsating during life, but they were extremely tortuous and difficult to trace. After death it was found that the circulation outside the abdomen had been carried on by means of communicating branches between the internal mammary and deep epigastric arteries, between the intercostal and deep and superficial epigastric arteries, and between the lower intercostal and superficial circumflex iliacs; all of which were greatly enlarged. The anastomosing branches were very tortuous, and ramified in every direction. One lateral branch, given off from the inner side of the epigastric artery, penetrated the umbilicus; and, running along the free border of the suspensory ligament of the liver, entered the longitudinal fissure of that organ, and anastomosed with a branch of the hepatic artery. Within the abdomen, the circulation had been carried on, as already mentioned, between the colica media branch of the superior mesenteric artery and the colica sinistra branch of the inferior mesenteric artery, including its sigmoid and hæmorrhoidal branches, and also between the upper lumbar

and ilio-lumbar arteries and the lower lumbar arteries and circumflex iliacs.

Last year a second case of abdominal aneurism, cured by compression of the aorta above the umbilicus, was communicated to the Society by Dr. Moxon and Mr. Durham of Guy's Hospital. A similar case, cured in the same manner by Dr. Heath, of Newcastle-on-Tyne, was referred to by Dr. Murray in a paper read by him before the British Medical Association at Dublin in 1867,* but the details of this latter case have, so far as I can ascertain, never been published. The same remark applies also to several other cases briefly referred to by Dr. Murray in his pamphlet on the rapid cure of aneurism by pressure, and some of these, moreover, were cases of compression of the aorta, not for aneurism of the aorta itself, but of vessels below its division into the iliacs.

Thus, the two cases published in the 'Medico-Chirurgical Transactions' are the only two cases of cure of abdominal aneurism by proximal pressure upon the aorta which have, as yet, been fully reported. These cases, indeed, may be considered to have sufficiently proved both the possibility of compressing the abdominal aorta above the umbilicus without causing serious injury to neighbouring organs; and, also, the fact that the circulation may be efficiently maintained through collateral vessels, even when the passage of blood through the abdominal aorta is shut off above the inferior mesenteric artery. Nevertheless, I venture to hope that the communication to the Society of a third case, also successfully treated upon Dr Murray's plan, may not be considered superfluous; more particularly as, in my case, some of the results of the compression applied over the aorta appear to have an interest apart from that belonging to the cure of the aneurism.

Christopher Fawcett, a strong-looking, well-nourished man, æt. 28, by occupation a warder in the House of Correction at Kendal, but who had previously been a sailor,

* 'British Medical Journal,' 1867, vol. ii, p. 287.

was admitted into the Middlesex Hospital under my care on May 20th, 1872.

When eight years of age he had been struck by lightning and had remained blind for three weeks afterwards, and at seventeen years of age he had suffered from rheumatism in the shoulders, knees, and ankles; but, in all other respects his health had been good until the commencement of the ailment for which he was admitted.

In December, 1868, being then an able-bodied seaman on board H.M.S. Princess Charlotte, stationed in China, whilst drawing water from alongside he suddenly felt something give way in his abdomen. This was followed by severe pain shooting downwards, and by faintness; both of which, however, shortly subsided. Two days later he became conscious of slight pulsation in the epigastrium, he felt weak and soon began to lose flesh. He was sent into hospital, where he remained five weeks, and was then invalided and sent home. Very shortly after his return to England he obtained his present employment as a warder. In December, 1871, he began to suffer from pain in the loins, creeping round towards the groins and testicles, and gave up work for some weeks, during most of which time he confined himself almost entirely to bed. During this period of rest he improved very much, lost the pain, thought himself well and resumed his duty; but, at the expiration of a short time, the pain in the back returned and presently extended to the legs, rendering him again unfit for work, and he was sent up to the hospital.

On admission he complained of pain in the abdomen as well as of almost constant aching in the loins, with pains shooting downwards to the groins and testicles, and sometimes down the thighs. The organs situated in the chest and abdomen were found to be normal. The urine was acid, had a specific gravity of 1020, and was free from albumen. The pulse was 56, the temperature 98°. The left radial and also the left femoral pulse beat more strongly than the right. The heart's action was r[illegible] and its [illegible] clean, but rather accentuated,

times felt with the impulse. A distinct but diffused pulsation, synchronous with the radial pulse, was both seen and felt above the umbilicus. On examination, a somewhat globular pulsating tumour, about the size of a large orange, was found in the abdomen immediately above the umbilicus. It extended more to the right than to the left of the median line, and beat forcibly with an expanding lateral as well as with a forward impulse. On deep pressure a thrill was also felt with the pulsation. On auscultation over the tumour no murmur was heard. Firm pressure over the aorta above the tumour, when the patient was sitting up in bed and inclining forwards, stopped the pulsation for the time being.

On the day after his admission the patient was seen by my colleagues, Dr. Henry Thompson, Mr. De Morgan and Mr. Hulke, who all agreed with me as to the nature of the tumour, and the propriety of endeavouring to effect a cure by the application of pressure upon the aorta above the aneurism, until the contents of the tumour should have coagulated. Mr. Hulke kindly undertook to apply and manage the tourniquet. The patient's bowels having been thoroughly cleared out on May 24th, the administration of chloroform was commenced at a quarter to three o'clock on the afternoon of May 25th. Ten minutes later Lister's tourniquet was screwed down between the tumour and the xyphoid cartilage, until pulsation ceased both in the tumour itself and in the femoral arteries. Vomiting, however, very shortly supervened, and after the pressure had been alternately relaxed and reapplied several times it was finally withdrawn, on account of the retching, at twenty minutes to four o'clock.

May 26th.—The patient had slept well and had passed urine abundantly. He complained of nothing but acidity on the stomach. His pulse was 79. The tumour was now more defined, extending two inches to the right, and one inch to the left, of the umbilicus. The impulse remained as before, but the tumour felt rather more solid; and a faint [illegible]mur, synchronous with the impulse, was heard at the

upper part of the tumour where the stream entered from the aorta.

27th.—Urine neutral, sp. gr. 1006, free from albumen. A large enema was given in the morning to empty the bowels, and at a quarter before seven in the evening, chloroform was administered by Mr. G. Everitt Norton. At the end of seven minutes the patient was thoroughly under its influence, and Mr. Hulke applied the tourniquet firmly over the aorta about two inches below the xyphoid cartilage, with the effect of at once arresting all pulsation in the tumour and in the femoral arteries. This pressure was maintained for a period of four hours with two brief intermissions. About eight o'clock the clamp had to be readjusted, which caused a momentary interruption of the pressure, when a wave accompanied by a thrill was felt to pass through the tumour. Again, about ten o'clock, at the expiration of three hours from the commencement of the operation, the instrument was intentionally withdrawn for a few moments in order to ascertain the effect which had been produced. A thrill was again perceived, but the tumour felt decidedly more solid, as though some coagulation of its contents had taken place. During the first hour, the pulse varied from 80 to 100, and the respirations from 36 to 48, in the minute; during the latter part of the time the pulse ranged from 100 to 120, and the respirations from 44 to 56, per minute. The breathing was very shallow and somewhat gasping. After the pressure had been maintained for some time there appeared marked lividity of the lower extremities which, as well as the lower half of the abdomen, became quite cold. The temperature taken between the first and second toes was 90°.

Sphygmographic tracings of [illegible] pulse taken from time to time [illegible] shewed, when [illegible] taken before [illegible] increased de[illegible] much shorten[illegible]

of the pressure at eleven o'clock was immediately followed by a marked change in the breathing, which having for some time been very short and shallow became at once much deeper and longer; the pulse resuming at the same time almost its normal force and frequency. There was still well-marked forward pulsation in the tumour, but the lateral expansion was much reduced. Three quarters of an hour after the withdrawal of the tourniquet, the patient vomited about half a pint of grumous, dark brown fluid. The pulse was then 104, forcible and of good volume; the respirations were 32.

28th.—The patient had throughout the night been almost constantly vomiting small quantities of brown fluid. It had been nearly four o'clock in the morning before he entirely recovered from the anæsthesia produced by the chloroform. He was much depressed, said he felt very bad, and complained of "pins and needles" in the feet and of a sensation of soreness in the stomach. There was a tender red bruise in the epigastrium at the spot on which the pressure of the tourniquet had been exerted. The pulsation in the tumour was undoubtedly decreased; the upward impulse being much less forcible and the lateral expansion only slight. The pulsation in the femorals was very feeble. The feet were numb, and the patient could but just feel when they were touched. Pulse 72. Temperature 99°. Urine acid, sp. gr. 1025, very considerably albuminous.

The vomiting continued more or less until June 1st. For two days the vomit contained altered blood, but afterwards it consisted of frothy, watery fluid, mixed with any liquid food he had taken. On May 29th the urine had a sp. gr. of 1030 and deposited a copious precipitate of lithates, but was free from albumen. The pulse has ranged from 60 to 68, and the temperature from 98·6° to 100°. The bruise and the feeling of tenderness in the epigastrium had disappeared. The patient still complained of numbness and pain in the right leg; the right limb was cold and livid, the left much less so. On May 31st pulsation

was distinctly felt in the left femoral and posterior tibial arteries, but none could be felt in those of the right limb.

The treatment consisted in the hypodermic injection of one sixth of a grain of morphia each day, and in the restriction of the patient's diet to milk with lime water or soda water, eggs, arrowroot, and beef tea. Strong beef-tea enemata were also administered during the two days that he was entirely unable to retain food. The legs were wrapped up in cotton wool and the lower end of the bed was slightly elevated in order to keep the feet in a raised position.

June 1st.—Patient had slept well; could now take an egg beat up in tea, or some toast and coffee, without being sick. Pulse 80; temperature 99·6°. Bowels had acted once. Urine sp. gr. 1015, alkaline, free from albumen. Patient said he felt very well and very hungry. The numbness was much decreased in the right leg which now felt warm, but the foot continued cold and livid. Pulsation, though quite distinct in the left femoral, could still not be felt in any of the arteries of the right limb. The pulsation in the aneurism had very greatly decreased; there was slight lateral expansion, but the forward impulse over the tumour was scarcely greater than over the aorta above it.

5th.—Patient complained of a burning sensation in the right foot, which had become warmer and less livid and was perspiring; the great toe alone remaining cold. There was distinct pulsation in both superficial pubic arteries, in the left superficial epigastric, and right circumflex iliac arteries, and also in the right femoral artery between Poupart's ligament and the middle of the thigh. The pulse in the left femoral appeared to be weaker than formerly, and scarcely any impulse could be felt in the aneurism.

8th.—A very feeble impulse could be felt two inches above the umbilicus, accompanied on auscultation by a faint blowing murmur. Pulsation was also felt in the dorsal artery of the right foot.

10th.—The patient was allowed to sit up for a short

time, but almost immediately after rising he experienced severe aching pain, accompanied by a sense of heat commencing in the loins and spreading forward to the crest of the ileum and downwards to the feet. On returning to bed he had a profuse sweating. A day or two later he was again allowed to sit up, and did so without experiencing any return of pain in the loins; but the right foot again became somewhat cold, and there was pain and tenderness in the great toe, which also presented a livid hue.

24th.—The pulsation in the tumour having decidedly increased again in force during the previous week, it was agreed in consultation with Mr. Hulke and my other colleagues to renew the application of pressure upon the aorta above the aneurism. The bowels were accordingly cleared out by an enema early in the morning of the 25th, and the usual precautions were taken to avert sickness from the chloroform. Mr. Morris, who had been present and had administered the chloroform from the end of the first hour on the previous occasion, again rendered us his assistance, and at half past eight in the evening the patient was brought thoroughly under the influence of the anæsthetic. Five minutes later Mr. Hulke skilfully adjusted Lister's tourniquet so as thoroughly to compress the aorta. The pressure was maintained for three hours continuously, with the exception of five minutes, during which it was intentionally relaxed a little before nine o'clock. Also, whilst the hand which held the tourniquet was being changed in position at a quarter past nine, a wave of blood was felt to pass through the aneurism. Immediately before the administration of chloroform was begun the radial pulse beat 79, and the respirations were 20 in the minute; both pulse and breathing being regular and quiet. Within ten minutes after the application of the tourniquet the pulse rose to 100, and the respirations to 40, in the minute; these latter, as well as the sphygmographic tracings of the pulse, being of the same character as during the former operation. Soon after the commencement of the pressure the right foot was observed [illegible] colder than the left. When the

tourniquet was removed at thirty-five minutes past eleven o'clock there was violent forward pulsation in the tumour, but no lateral expansion could be detected; no murmur could be heard and the tumour felt firmer and more solid. One sixth of a grain of morphia was injected hypodermically half an hour after midnight.

26th.—Patient had slept most of the night, but had been sick several times. The matter vomited consisted only of clear, greenish fluid and presented no appearance of blood. Pulse 84, respirations 22. Three ounces of acid urine had been passed, sp. gr. 1025, containing one fourth, by measure, of albumen and a large number of casts, chiefly hyaline, but a few granular and a few others with epithelial cells adhering to them. The impulse of the tumour was much lessened, and no pulsation could be detected in either femoral artery. A rather large artery was found beating on the right side of the epigastrium.

During five days the sickness continued to recur at frequent intervals. At first it was quite uncontrollable, and the patient felt so weak and faint that it was necessary to give him an enema of beef tea, half an egg and a table-spoonful of brandy every three or four hours.

28th.—He became slightly jaundiced, but it only lasted two or three days. The urine continued albuminous for two days, and then became normal again. Both feet remained cold and livid for several days, but the patient was able to move them freely. The pulsation in the aneurism continued to diminish and was exclusively forwards. The tumour felt more solid and its margin became more defined, especially on the right side.

July 1st.—The impulse in the aneurism could not be seen and could scarcely be felt. The patient was allowed to sit up on July 10th, and was well enough to be discharged on July 14th, in consequence of the necessity of closing the hospital for repairs. A day or two later he returned home to Kendal.

On September 20th the patient, in accordance with my request, made when discharging him in July, returned and was readmitted into the hospital. He stated that since

his leaving it he had had no return of pain in the back or groins, but that for some time after his return home he had frequently at night felt "pins and needles" in his feet, and more especially in the right foot, compelling him to hang his legs over the bedside. He had also, after taking walking exercise, suffered from pain in the calves and from a sensation of coldness in the feet. He was carefully examined by Mr. Hulke, Mr. De Morgan, Mr. Morris and myself. No pulsation was found in the seat of the aneurism, neither was there any distinct tumour remaining; but immediately above the umbilicus, a little to the right of the median line, there was an undefined hardness which appeared somewhat movable. Posteriorly, a slight degree of dulness on percussion was found over a limited area of the lower ribs on the right side. On auscultation over the left loin an indistinct pulsation was heard, accompanied by a humming or buzzing sound. No pulsation nor aortic sound could be detected in the aorta from an inch above the umbilicus downwards. Below the epigastrium a murmur, unaccompanied by any impulse, was heard, which was probably communicated from neighbouring arteries. No pulsation was found in the femoral, popliteal, or anterior tibial arteries; but there was feeble pulsation in the left posterior tibial artery, and, Mr. Morris thought, in the right also. Pulsation was felt, and had been noticed by the patient himself, in the seat of the deep epigastric arteries on both sides. A superficial vessel about the size of the radial, was felt beating in the walls of the abdomen, about two inches above the umbilicus. Small vessels were also felt pulsating near the crest of the ileum and the trochanter major. No pulsation could be detected about the buttocks, but Mr. Norris found with the finger in the anus three or four large arteries of the size of crowquills beating very distinctly. The lower limbs were somewhat emaciated, but healthy in colour and perfectly warm.

Mr. Noble, of Kendal, who had originally sent up the patient to the Middlesex Hospital, wrote me word later in the autumn that the man was in perfect health and had re-

turned to his duty as warder in the House of Correction. All trace of pulsation in the site of the aneurismal tumour was obliterated.

It will have been observed in the history of this case that the pulsation in the aneurism continued for a considerable number of days after the last application of pressure to the aorta. In this respect it resembles Dr. Moxon's and Mr. Durham's case, but differs from Dr. Murray's. In the latter case, although some very slight pulsations were felt when the tourniquet was removed, the tumour had become perfectly pulseless by the evening of the same day, and every indication of pulsation in the aorta below it was found to have disappeared.* In Dr. Moxon's and Mr. Durham's case, on the contrary, pulsation in the aneurism continued for a month.†

Another fact in the history of the present case, deserving of particular notice, is the gradual relapse after the period of favorable progress which followed the four hours compression of the aorta on May 27th. For some days the impulse in the aneurism had been gradually subsiding, until on June 10th it was scarcely perceptible, and the patient was allowed to sit up for half an hour. Then it again began to increase and on June 24th it seemed obvious to us that, unless the pressure were reapplied, the tumour would soon return to its original condition. It seems to me now very possible that if the patient had been kept perfectly at rest for some time longer, the consolidation, which had partially taken place during the latter days of May, might have continued to progress, and the cure might have been completed without any further compression. But I was then unacquainted with the details of Dr. Moxon's and Mr. Durham's case, and knew only the history of Dr. Murray's, in which very rapid coagulation took place; and, therefore, as a stream of blood still continued to pass through the aorta, I did not expect the cure to be completed without again having recourse to compression. It would

* Loc. cit., p. 20.

† 'Med.-Chir. Trans.,' vol. lv, p. 218.

now, however, appear certain that the process of cure in these cases, by coagulation of blood in the sac of the aneurism, is not necessarily a rapid process, but may go on so slowly that the passage of blood through the aneurism may not cease for many days, nor in some cases until after the lapse of weeks.

The results, in the present case, of the compression over the aorta, which appeared to me to have an interest apart from that belonging to the actual cure of the aneurism, were the effects produced, first upon the general circulation, and, secondarily upon particular organs.

The effects upon the pulse were very remarkable, both as felt by the finger and shown by the sphygmograph. It became much more frequent, whilst the short upstrokes and rounded summits presented by the sphygmographic tracings indicated diminished expansion and delayed collapse of the artery. At the same time the breathing became shallow and the respiration frequent.

These characters of the pulse and respiration supervened almost as soon as the pad of the tourniquet was screwed down, and disappeared quite suddenly the moment the pressure was removed.

The organs secondarily affected by the results of the pressure upon the general circulation were the stomach and kidneys. Hyperæmia of these organs from the forcible distension of their capillaries, consequent upon the sudden shutting off of the ordinary supply of blood to the arteries below the compressed portion of the aorta, was undoubtedly the cause of both the hæmatemesis and the albuminuria which followed the operation. The prolonged and for some time uncontrollable vomiting, which on both occasions followed the operation, was likewise, in all probability, partly due to gastric hyperæmia, for it can scarcely be regarded as having proceeded altogether from the chloroform. It is, however, very probable that pressure on the solar plexus of nerves may have contributed to the result.

The occurrence of such symptoms appears to me to suggest, that the intense arterial distension, produced by the

treatment, might be attended with serious danger in persons suffering from any kind of organic disease.

The three patients whose cases are now fully recorded were all healthy young men, and in each of them the aneurism seems to have arisen from a mechanical cause. Had any one of them been the subject of arterial atheroma, of valvular disease of the heart, or of disease of the lungs or kidneys, there appears to me to be grounds for apprehending that the case might have terminated less favorably.

ANALYSIS OF OBSERVATIONS

ON THE

TEMPERATURE, PULSE, AND RESPIRATION,

IN

PHTHISIS AND ACUTE TUBERCULIZATION OF THE LUNGS.

BY

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THE pyrexia of phthisis has been known from the earliest periods of medical observation to constitute one of the most important features of the disease, and during the greater part of the present century, it has, with more or less frequency, been made the subject of thermometric observation.

The changes in the temperature in this disease are, however, subject to such great irregularities, that it was long before any general description was given of the variations observed, until the publication of Jochmann's thesis,* and of Wunderlich's work on the thermometry of disease.

Jochmann's descriptions so nearly comprehend the chief variations in the types of fever which occur in phthisis, that as his paper appears but little known in this country, it may perhaps be desirable to give a brief abstract of these, since I shall have again to refer to them.

* 'Beobachtungen über die Körper-Wärme in Chronischen Fieberhaften Krankheiten,' 1853. Conducted under the direction of Prof. Traube, of Berlin.

TYPE I.—The temperature scarcely exceeds the normal standard, but that of the evening is slightly higher than that of the morning.

(*a*) Occasional evening exacerbations of a higher range.

(*b*) Morning temperature occasionally below the normal.

TYPE II.—The morning temperature normal or subnormal. The evening temperature above normal.

Var. A. The evening temperature only slightly above normal.

(*a*) On many days the evening temperature normal.

(*b*) The morning temperature occasionally above normal; the evening temperature constantly so.

Var. B. Evening temperatures considerably above normal, while the morning temperature remains normal and nearly constantly so, and may be subnormal. This is the "intermitting" type which may be quotidian or tertian.*

TYPE III.—The temperature both of the morning and evening considerably above normal.

(A) The temperature highest in the evening, but very variable, the morning temperature though above normal, more regular.

(B) The temperature highest in the mornings.

(C) The temperature above the normal morning and evening, but reaching its maximum at midday.

Jochmann, however, points out that all these types may alternate with one another in a single case; the most persistently maintained temperatures being, however, the first and second classes as a whole and varieties A and C of

* To these Wunderlich adds a duplicated quotidian in which two exacerbations occur in a single day, an observation formerly made by Laennec, Forbes' 'Transl.,' 346, and by Andral, 'Clin. Med.,' iv, 164. Laennec observed rigors to precede each exacerbation. Professor Lebert has also noticed this form, 'Deutsch. Arch. Klin. Med.,' xi, 91. The tertian type is rare. Jochmann only observed it in one case, in which the course was somewhat interrupted by slight exacerbations. A case of this nature will be subsequently quoted.

Class III. Those which are least persistent are var. B, Class III (morning temperature the highest), and var. B, Class II (high evening and normal morning temperatures). The type of high morning and evening temperatures co-existing is often interrupted by normal morning temperatures.

Nearly all the observations hitherto recorded have, however, been made on limited numbers of cases, each case being taken as the type of a class. Finding that the great irregularity in temperature rendered exact observation for therapeutic purposes uncertain, I wished to ascertain the predominant changes in different groups, and determined, therefore, to analyse a larger number of cases. The observations on which this analysis is founded have been made by a succession of gentlemen holding the office of Physicians' Assistants in University College Hospital, and the high position obtained by many of them gives the best guarantee for the accuracy of these data. To some of them I am indebted for notes furnished at my request of cases not immediately under my own charge, and I have also to thank my colleagues (Sir W. Jenner, Dr. Hare, and Dr. Reynolds), for permission to supplement some of my notes from their case-books. The large majority of cases have been, however, those under my own care, and the observations analysed have in such cases been made by gentlemen acting as my assistants. I have also to thank Dr. Charles H. Carter for some valuable assistance in this analysis.

In one respect the fact that the observations were conducted solely in the mornings and evenings, gives to these results only an approximative value, inasmuch as during the remainder of the twenty-four hours considerable variations in temperature may occur, but they may, I think, be taken as representing fairly the results of a comparison of the temperatures at these periods, in groups of cases, when the observations are conducted in the manner ordinarily pursued in clinical observation, and if they in some respects show the need of a more extended enquiry respecting the course of the temperature during the remainder of the

twenty-four hours they will, though in a subsidiary manner, be, I trust, not without their value.

When this paper was drawn up by me no general statement of this kind had, as far as I am aware, been previously attempted. Since it has been written, an analysis conducted on somewhat similar principles, has been published by Professor Lebert.* As the method of analysis pursued by Professor Lebert differs somewhat from that which I have followed, I have thought it best to leave my own unchanged. I shall, however, frequently have occasion to refer to Professor Lebert's paper in illustration of the results of my own analysis.

I must call attention also to another fact, viz. that the cases here analysed have been instances of more or less distinctly advanced disease, admitted to a general hospital on account of its acuteness, its extent, or for recent aggravation, and that those not ending fatally have been discharged when the more urgent symptoms have been alleviated. The observations thus recorded represent therefore the symptoms of a group of severe cases, and they thus afford no information respecting the earlier stages or the periods of comparative quiescence. They have also with the exception of a few of the cases of acute tuberculosis been nearly all instances of phthisis occurring in the adult.

I have thought it advisable to analyse the records of the pulse and respiration taken simultaneously with the temperature in their relation to it and to one another. The latter phenomena have already been made the subject of a complete and exhaustive paper by Dr. Edward Smith, published in the 'Transactions' of this Society.† As Dr. Smith's observations had the greater advantage of being carried on continuously during the whole of the twenty-four hours, I should have thought it undesirable to publish my own analysis except so far as relates to the comparison of the pulse and respiration with the temperature, were it not also that the number of cases here investigated shows some

* 'Deutsch. Archiv. Klin. Med.,' Nov., 1872.

† 'Med.-Chir. Trans.,' vol. xxxix.

results not yet presented to the profession in this form. The cases thus analysed are eighty in number, and the results are expressed in a tabular form. The numbers of the cases vary somewhat in the different tables, as I found it necessary to exclude some, according to the subject of investigation, where the observations on the points in question were not sufficiently numerous to afford accurate data, and this is especially the case where averages have been taken. As, however, the results are for the most part expressed in percentages, this will not, I trust, prove a source of any difficulty to those who may think them worthy of perusal.

I have divided the cases thus analysed into five classes, corresponding with some exceptions, not to their thermometric but to their clinical features, and I have thought it desirable to maintain this distinction as showing the variations which may occur in the temperature in groups of cases which are otherwise apparently similar.

The classes which I have thus formed are—

A. Fatal cases, viz.—(1) Acute tuberculosis. (2) Acute pneumonic phthisis, including under this category cases of less than eighteen months' total duration as far as could be judged from their history, and the majority of which ran their course in less than a year or nine months. (3) Chronic phthisis, defining these as more than eighteen months in duration, many of them having been ill for several years. (This distinction is of course arbitrary, but, without going into details on this point, I would add that it was confirmed by post-mortem results, showing either pneumonia or recent granulations or freshly formed cavities in the former, and indurations, often of considerable extent, in the latter class.)

B. Non-fatal cases have also been arbitrarily divided by me into two classes, those with a high and those with a low temperature. Those, for the sake of brevity, I shall speak of as "High Temperatures" and "Low Temperatures."

It is possible that the numbers thus represented in the different classes may yet fail to exhibit all the phenomena

which might be observed in larger groups. I think it especially necessary to call attention to this fact in relation to acute tuberculosis, since the cases which I have met with present only the severely pyrexical type, and it must be considered as certain that another group must be constructed in this affection (of which, however, I have only seen one illustration when the data were not sufficiently numerous to be included in this analysis), in which very low ranges of temperature occur, this being either scarcely febrile or below the normal standard; and probably also intermediate gradations may be found when the pyrexia is moderate.*

The facts I have analysed in the different classes relate chiefly to the following subjects:

I. The maximum temperature observed.

II. The averages of the morning and evening temperatures.

III. The frequency of normal or subnormal morning and evening temperatures.

IV. The comparison of the temperature of the morning and evening.

V. The remissions and exacerbations occurring from evening to morning and from morning to evening.

VI. Circumstances influencing the pyrexia.

I have also analysed the maximum, the minimum, and the mean frequency of the pulse in the morning and evening as compared with one another, with the temperature, and also with the respiration, at these periods; the rate of the respiration at the same periods as compared with the temperature; and the pulse-respiration ratio as compared with the temperature. The degrees of temperature are expressed in Fahrenheit's scale.

§ I. *The temperature attained* varies considerably in different cases of phthisis. The general range, at least in one class of acute tuberculization, may be higher than in that

* In illustration of this fact I would especially refer to Dr. Gee's article, "Tubercular Meningitis," 'Reynolds's System,' vol. ii. Dr. Ringer, 'On the Temperature of the Body as a means of Diagnosis in Phthisis and Tuberculosis,' several cases. Dr. Finlayson, 'Glasgow Medical Journal,' 1869, ii. Steffen, 'Klinik der Kinderkrankheiten,' p. 272 *et seq.*

of all the other forms, a fact also observed by Professor Lebert, but another group of this variety of the disease must, as before stated, certainly be formulated, in which no such preponderance exists over even those presenting the lowest scale of temperatures.

The higher ranges of temperature in this form are as a class almost equalled by some cases of acute phthisis, and by some, not ending fatally, presenting high temperatures.* Thus, of the cases here analysed (see Table 2) a maximum temperature of 104° was exceeded by 62·5 per cent. in acute tuberculosis, by 36 per cent. in acute phthisis, by 37·5 per cent. among the high temperatures not ending fatally, by 11 per cent. of the cases of chronic phthisis, and by none of the cases presenting low temperatures not ending fatally.† Temperatures exceeding 105° were, however (with the exception of two single observations, occurring respectively in cases of acute and chronic phthisis), exclusively found among the cases of acute tuberculosis (25 per cent., see Tables 4 and 5). The maximum temperature which I have met with has been 106·7°, but it only occurred on one occasion in a case of acute phthisis.‡ Wünderlich says that hyperpyrexial temperatures may occasionally occur towards death, but of this I have not seen an instance.§

In all the fatal cases, with the exception of eight, the temperatures were higher than in the non-fatal, but a lower

* Lebert found in 22 cases the mean evening temperature of acute tuberculosis was as follows:—In 2 not exceeding 100·4°; in 8, including those last mentioned, not exceeding 100·2°; in 9 more than 103·4°; in 4 from 103·4° to 104°; in 1 from 104° to 105·5°. The maximum of these cases was, however, high.

† This distinction is of course arbitrary; the mere existence of such temperatures would in this arrangement have placed these cases in a different class.

‡ I have in another case, not here tabulated, met with a maximum temperature of 106·5°. Lebert does not appear to distinguish this variety as distinct from acute tuberculosis.

§ Lebert records one case when the temperature rose between 107° and 108° F. He remarks that hyperpyrexial temperatures at the close of life are most commonly found in acute tuberculosis.

range of temperature existed in the cases of chronic phthisis than in the others. Thus, of cases where the temperature did not exceed 103°, 25 per cent. were found in acute tuberculosis, 28 per cent. in acute phthisis, none such occurred among the high temperatures not ending fatally, 38·8 per cent. were found in chronic phthisis, and 90·3 per cent. among the low temperatures not ending fatally; while, below 102°, only three cases were found in acute phthisis,* and also in the cases of chronic phthisis, and 89·3 per cent. in those with low temperatures. (See Tables 1 and 2.)

The chronic cases ending fatally present in their temperatures in certain instances an approximation to those observed in the more acute forms; and this will be readily understood from the fact that their progress is frequently by means of a succession of acute or subacute attacks which in their final aggravation are often largely pneumonic in their character, and by which probably the high temperature is determined; such pneumonia being observed, though often in an indurated form, in all those where the temperature exceeded 102°. In one case only of this class did the temperature not exceed 100°. It was in a female, æt. 43, with a history of past phthisis, and in whom death was due to bronchitis, in emphysematous lungs, in which, in addition to old indurations, recent grey granulations had developed. In five only, however, did any large proportion of the observations exceed 102° (40, 48, 56, 60, and 84 per cent. of the observations in each case), and all these were characterised by old indurations and recent extensive pneumonia, combined with recent tuberculization. In two cases of "low temperatures not ending fatally," an evening temperature of 103·6° and a morning temperature of 103° were each reached on one occasion only, but the average temperatures of these two cases were respectively—mornings, 98·9° and 99·9°; evenings, 100·2° and 99°. In two others a temperature of 102·8° was attained by each on two occasions only, and the average

* In all these there was much pulmonary induration, pointing to a probably longer course than that assigned by the patient.

morning and evening temperatures of these cases did not exceed 99·4° and 100°.

The class of "high temperatures not ending fatally" resembles in this respect a large proportion of cases of "chronic phthisis," that they all presented a pneumonic attack supervening on pre-existing phthisis. Their average temperatures (see Tables 3, 4, and 5), are lower than in the other acute cases; but this is due to several of them undergoing, while under treatment, a marked improvement and a subsidence of the pyrexia. Their maximum temperatures are, however, nearly on a par with those of the acute forms.

§ II. Considerable differences are apparent in the *averages* of both the morning and evening temperatures observed in the individual cases in each class. (See Table 5.) These averages are higher in the larger proportion of cases of acute tuberculosis, acute phthisis, "high temperatures," and chronic phthisis, than in those of "low temperatures." In the latter the larger proportion do not show an average either for the morning or evening exceeding 100° F.; but in the former there are a large number both of morning and evening averages exceeding 101°, none of which are found in the latter class.

This may be shown on analysing Table 5. Thus, in each class the percentage of the cases, when the *average* morning and evening temperature did not exceed 100°, and the cases where these exceeded 101°, may be thus represented.

Class.	Average morning not exceeding 100°	Average evening not exceeding 100°	Average morning exceeding 101°.	Average evening exceeding 101°.
Acute tuberculosis	12·5	0	50	87·5
Acute phthisis	28	8	44	60
High temperatures	62 5	12·5	25	37·5
Chronic phthisis	34·3	27·7	22·2	49·9
Low temperatures	99·9	85·6	0	0

Averages of evening temperatures exceeding 102° were found in 75 per cent. of the cases of acute tuberculosis, in 24 per cent. of the cases of acute phthisis, in 11·1 per cent. of chronic phthisis, and none occur among the classes of "high" and "low temperatures" not ending fatally. The averages of the evening temperatures are, as a rule, considerably higher than those of the morning.*

The class of "high temperatures" shows an even larger proportion of morning temperatures, which on the whole are comparatively low, than those of chronic phthisis. This may in part be due to the conditions already indicated, as observed in these cases; possibly, also, to the comparatively small number of cases observed. It will be, however, again alluded to.

The average of the whole series of morning temperatures in any given case in all classes may, indeed, be found within normal limits of temperature (not exceeding 99°). The same fact may be observed for the evening averages, but with comparative rarity, except in the class of "low temperatures." (See Table 5.) Morning temperatures with an average within the normal were not, however, observed in any of the cases of acute tuberculosis occurring under my observation, but they have been met with by others.† They are more common in chronic phthisis and in the class of "low temperatures" than in the acute form, but they were met with in the latter variety in three out of twenty-five instances.‡ One of these presented on the whole a low scale of temperature throughout, exceptional among this class.§ In the

* M. Lebert found that in 10 cases only out of 149 was the mean evening temperature between 98·6° and 100·4° (2 of these were acute cases). In 22 per cent. the average evening temperature ranged from 100° to 102° (3 acute cases). In the greater number, or nearly one half, the mean evening temperature varied from 101° to 103°; and these higher ranges were nearly equally distributed among the acute and the chronic cases.

† Cf. Ringer, l. c., obs. pp. 79, 87, 89; Finlayson, l. c., obs. ii and viii; Steffen, l. c., obs. xxxi.

‡ Cf. Table V. In chronic phthisis they occurred in 5 out of 15 cases, or 27·7 per cent.; in the "low temperatures" in 11 out of 21 cases, or 52·3 per cent.

§ The reason for putting this case among the acute class was the history of the patient that his disorder was only of

other two the low average of the mornings was the result of the marked remissions which ensued after the evening exacerbations, and which may fall to subnormal degrees.* The five cases when this was observed in chronic phthisis were, without exception, those with a comparatively low range of temperature throughout. A maximum of 103° was only exceeded in one, a maximum of 102° in three, while in the fifth this did not exceed 100°. Subnormal morning temperatures, corresponding to more marked remissions, also occurred in all but in one out of the five. Occasional morning temperatures with a maximum exceeding 103°, and in two with a maximum exceeding 101°, were observed in these cases. In the other two the morning temperature never exceeded 99°, and was frequently subnormal. Considerable remissions also occurred in all these cases (in one observation 6°, in one 5°, in one 4°, in two 3°, in one 2°).

In the cases with "low temperatures," when the average morning temperature was normal, this was almost always accompanied by a low evening and a nearly natural, or occa-

an old large anfractuous cavity in the lung, and albuminoid disease of the spleen, leading to the suspicion that the disease was of older standing. The maximum morning temperature was 99·8°, the maximum evening 101°. The average morning was 98·8°, the average evening 99·7°.

* In one of these the maximum remission amounted to 8·8° (the highest I find recorded). The average of the remissions from evening to morning was 3·9°. High morning temperatures, however, occurred occasionally; the maximum morning was 102·8°, the maximum evening 103·2°. The average morning was 98·6°, the average evening 100·7°. Subnormal temperatures occurred in the morning in 7, and normal in 2, out of 25 observations. The morning temperature was, however, higher than that both of the preceding and following evenings on six occasions respectively; and in 2 of the latter the evening temperature was on one occasion normal and on another subnormal, a fact which accounts for the comparatively low average of the evening temperatures. The other case was also an acute one (coming on within a month after confinement of a premature child), with marked remissions from the evening to the morning, the latter being constantly at or below normal, only once rising to 99·8°, while the maximum evening temperature was 103°, and the average 101°; the maximum remission being from evening to morning 4·6°, and the average of these remissions 2·7°. The lesions present were recent grey pneumonia of one apex with soft granulations in it, grey granulations of the opposite [illegible], tubercular peritonitis and tubercular ulceration of the intestines.

sionally a subnormal morning temperature. In one only was a morning temperature above 100° observed (102°), and in two only did the evening temperature ever exceed 100° (102° and 103°). These cases, therefore, came for the most part within Jochmann's first type, though this was not always regularly maintained, and the morning temperatures in eight of the eleven were occasionally higher than those of the evening.*

Averages of the evening temperatures not exceeding 99° were met with by me only in cases of chronic phthisis and the class of "low temperatures." They have, however, been met with in cases of acute tuberculosis.† In the two cases of chronic phthisis in which they occurred the low average was due to subnormal temperatures preceding death. In the class of "low temperatures," they appeared to be almost invariably due to a general low range of evening temperature (which in none of these exceeded 100°), together with occasional remissions from the morning to the evening; which, in one of these cases to be hereafter alluded to, completely inverted the regular order (the morning being always higher than the evening), and in three others varied from 30 to 40 per cent. of the observations.

§ III. The total number of cases in which during twenty-four hours a *normal temperature* was observed during some part of the case, is seen in Table 2. The proportional frequency of *normal and subnormal morning and evening temperatures*, both as to the cases and the relative number of observations in which they occurred, is shown in Table 3. Normal and subnormal morning temperatures are there seen to be more common than the

* M. Lebert says that in 33·3 per cent. of all his cases the mean morning temperature only exceeded the normal in exceptional instances. In one third of his cases the mean morning temperature was between 98·6° and 101·3°. The cases with high mean morning temperature only amounted to 14 per cent. of the whole number. The morning temperature, therefore, tends to be low in all varieties of the disease. High morning temperatures occasionally, however, occurred, in one instance exceeding 105°.

† See Steffen's obs. xxxi, before quoted.

same phenomena in the evening;* but a fair proportion of the latter is observed in all the classes except that of acute tuberculosis. (It must, however, again be recalled that they do occur in this class also.) It is, however, only in very exceptional instances that they occur in any case in any considerable number of observations. The conditions of such occurrence will be alluded to hereafter.†

§ IV. In *comparing the temperatures of the morning and evening* it will be found that the maximum temperatures, as a rule, are usually attained in the evenings, and therefore the average temperature is in the majority higher for the evening than for the morning. A fair proportion of exceptions exist, however, to this. In 8 cases out of 80 the maximum temperatures observed in the morning and evening were equal,‡ and in two of these the averages of the morning temperatures throughout the case were higher than those of the evening. In 19 more or 23·7 p. c. the maximum morning temperature was higher than the maximum evening observed.§

* M. Lebert found the *minimum* morning temperature below 96·8° in 28 per cent. of his cases; and only in 4 per cent. (6 only of 149 cases) did the morning minimum range from 99·5° to 103·5°. The morning maximum was, however, subnormal in 9 only of 149 cases; in two fifths it ranged from 99·5° to 101·3°, and in one half from 101·3° to 104·9°.

† M. Lebert found a minimum evening temperature not exceeding the normal in one third of his cases.

‡ One of these cases occurred in acute phthisis, 3 in chronic phthisis, 1 among the "high temperatures," 3 among the "low temperatures."

§ Of these 1 case occurred in acute tuberculosis, 6 in acute phthisis, 3 in chronic phthisis, 1 among the "high temperatures," and 8 among the "low temperatures."

Maximum morning temperature exceeded maximum evening by 2° in 1 case.
" " 1° in 4 cases.
" " 1° to 0·5° in 6 cases.
" " 0·5° to 0° in 8 cases.

Total 19 cases.

morning temperature exceeded average evening by 2° in 1 case.
" " 1° in 2 cases.
" " 0·5° to 1° in 1 case.
" " 0·5° to 0° in 6 cases.

Total 10 cases.

temperature exceeded average morning by 1° in 2 cases.
" 0·5° to 0° in 3 cases.

Total 5 cases.

In 10 of these the average of the morning temperatures was greater than, in 4 equal to, in 5 less, those observed in the evening. In two cases more, though the maximum morning temperature observed was less than the maximum evening, the average of the morning temperatures was greater than that of the evening,* and in another of the same class the averages were equal.†

High morning temperatures are observed, therefore, in a large number of cases of phthisis, and in nearly all the classes, with the exception of those of "low temperatures"; the proportion of observations in which 103° is exceeded is nearly equal, amounting from 50 to 52 per cent. of the whole number of observations. (See Table 4.) A larger number, however, of cases present a high average of their individual morning temperatures in acute phthisis than in any other class. For, of average morning temperatures exceeding 102°, there only occurred 1 case out of 8 (12·5 per cent.) in acute tuberculosis, while 6 out of 25, or 24 per cent., occurred in acute phthisis, and none in the other classes. The absolute number of cases in which an average of 102° was exceeded was thus in acute phthisis as great in the morning as in the evening, being in each 6 or 24 per cent. of the 25 cases examined, a proportion not found in any of the other classes. (See Table 5.)

Morning temperatures exceeding 104° were only met with in the classes of acute tuberculosis, acute phthisis, and the "high temperatures," in the relative order of 25, 15, and 12 per cent. of all the observations in these classes. None were observed in chronic phthisis and in the class of "low temperatures." ‡

* Both occurred in "acute phthisis."

† Occurred among the "low temperatures."

‡ Morning temperatures exceeding 103° were found in 4 out of 8 cases of acute tuberculosis (50 per cent.), in 13 out of 25 cases of acute phthisis (52 per cent.), in 9 out of 18 cases of chronic phthisis (50 per cent.), in 2 out of 8 cases of "high temperatures" (25 per cent.), and in 1 out of 21 cases of "low temperatures" (4·7 per cent.). Morning temperatures exceeding 104° were found in 2 out of 8 cases of acute tuberculosis (25 per cent.), in 4 out of 25 cases of acute phthisis (16 per cent.), and in 1 out of 8 cases of "high temperatures" (12·5 per cent.). The proportions of the number of CASES in

The fact that the morning temperatures are frequently higher than those of the preceding or following evening has been noticed by most writers on the thermometry of phthisis, but the extent to which this may prevail has been but little insisted on.* Fournet,† indeed, stated that morning pyrexia was more common in the earlier than in the later stages of the disease, but my observations hardly confirm this opinion.

Out of 75 cases there were only seven which at some part of their course showed no rise of temperature from the evening to the ensuing morning, and only 6 had no remissions from the morning to the ensuing evening. The morning temperature was therefore, occasionally, higher than that of the preceding evening in 90·7 per cent., and higher than that of the succeeding evening in 92 per cent., of the whole number of cases. (See Table 6, Appendix.)

The occasional existence of higher morning than evening temperatures within pyrexial limits was pretty evenly distributed through all the classes of cases with the exception of cases of acute tuberculosis.

	Acute tuberculosis; 8 cases per cent.	Acute phthisis; 24 cases per cent.	High temperature; 8 cases per cent.	Chronic phthisis; 15 cases per cent.	Low temperatures; 20 cases per cent.
Morning occasionally higher than previous evening ...	62·5	87·5	100	100	90
Morning occasionally higher than following evening ...	75	87·5	100	100	85

(See also Tables 7, 8, 9.)

This fact is also not unfrequently observed at temperatures within a normal range, when these occur in any part of the course of the case; but, even within pyrexial limits

which these were observed correspond, therefore, pretty closely with that of the observations in each case.

* Traube, 'Symptomen der Krankheiten der Respirations und Circulations Apparats,' p. 77, has noticed the relative frequency of this type of fever in phthisis. It has also been observed by Lebert, and has been termed by these authors the "typus inversus."

† "Rech. Clin. sur l'Auscultation des Organes Respiratoires," 1839.

a very large proportion of the whole number of observations may be found to present this peculiarity. (See Table 6.) In one case, in a series of observations extending over twenty-three days, the morning temperature was constantly higher than the evening, with the exception of three occasions when it was identical with that of the previous evening, and one occasion when it was the same as that of the following evening, these being within a normal range; and on seventeen out of these twenty-three days the temperature was pyrexial, though on a low scale.* The same phenomenon is, however, observed in all classes of phthisis, and may, though comparatively rarely, be present for many days in succession. On one occasion this was noticed during eleven days successively in a severely pyrexial case of acutely tubercular pneumonia, where it occurred in 23 out of 58 observations. In another case of pneumonia supervening on chronic phthisis it was noticed in 21 per cent. of 33 observations. Usually, however, it occurs irregularly throughout the case, but sometimes, as already remarked in a case of acute tuberculosis, on nearly alternate days.

I think it possible, from a limited number of observations, that in some cases, when the morning temperature exceeds that of the succeeding evening, this may be occasionally due to an intervening midday exacerbation,

* This case, a female, æt. 43, presented no peculiarities with the exception of an enlarged heart with a mitral murmur, the result of a previous attack of acute rheumatism. Cough had existed for years. There was emphysema, consolidation of both apices, and dulness of the left base with râles at both bases. There were also present some œdema of the ancles, without albumen in the urine; enlargement of the liver and diarrhœa, to which she had been subject for years, but which had no apparent effect on the temperature. The pyrexia subsided, and the dulness at the left base disappeared, though some râles remained. The morning temperature within pyrexial limits exceeded the previous evening in 70 per cent., the subsequent evening in 74 per cent. of the observations. In 52 per cent. of the whole number the morning was pyrexial, the evening temperature normal. The maximum morning temperature was 101·4°, the maximum evening 99·8°. The average of the morning temperatures was 99·5°, that of the evenings 98·6°.

The maximum exacerbation from evening to morning = 2° average 0·9°.

„ remission from morning to evening = 2° average 0·9°.

but my cases have not permitted me to make any accurate analysis of this point, as they contain very few midday observations.* This explanation is not, however, applicable to those cases where the morning temperature is higher than that of the preceding evening, in which a true inversion of the order of exacerbations appears to take place.†

* M. Lebert, loc. cit., also directs attention to this feature, though his cases are for the most part analysed like mine, solely with regard to the morning and evening temperatures. He, however, points out that the temperature may apparently fall from the morning to the evening, but that the chief fall may occur from morning to noon, and a slight rise occur towards evening. Thus, the fall from morning to noon may be 3·7°, yet the fall in the evening may only be lower than the morning by 1·4° fall. Again, when the temperature in the evening is higher than the morning, the greater part of the rise may be from the morning to noon, and a slight rise only towards evening. The temperature in phthisis, moreover, frequently oscillates considerably within a few hours of the same day. Thus, the difference between the morning and evening may be 2·3° fall, while the difference between the means of the maximum and minimum of repeated observations made in the twenty-four hours may be less than 0·2° fall.

† The course of the temperature in these cases also shows considerable variation. In one case of acute phthisis where out of 42 observations the morning temperature was on 19 occasions higher than on the preceding evening, and on 13 higher than on the succeeding evening, the following variations were observed:

(*a*) Temperature in morning lower than preceding evening, fell to following evening, but rose next morning.

(*b*) Morning temperatures then higher than either preceding or following evening for four days, but broken by a rise on the fifth evening.

(*c*) Temperature in morning higher than previous evening, rose to next evening, and fell to following morning.

(*d*) Temperature fell from evening to morning, fell to next evening, rose to following morning, and rose to next evening; fell to next morning, and fell further to next evening; all these temperatures being febrile.

In another case where out of 58 observations the morning temperature was on 20 occasions higher than on the preceding, and on 23 higher than on the following evening, the following variations were observed in respect of the first class:

Morning temperature higher than preceding evening, fell to following evening in . . .	14	1 normal, 1 subnormal, 12 pyrexial.
Morning temperature higher than preceding evening, rose to following evening in . . .	4	
ning temperature higher than preceding evening, mained equal to following evening in . .	2	
	20	

In a large proportion of all cases of phthisis the temperature of the morning, when febrile, is occasionally found to equal that either of the preceding or following evening, and this equality is also observed within the normal range in cases with low degrees of pyrexia. This, however, only occurs in each case, as a rule, in a limited number of observations, and it is seldom continued, though it is recorded in two cases as lasting during forty-eight hours.

The subjoined Table exhibits the number of cases in which occasional equality between morning and evening febrile temperatures was met with in 75 cases. In addition to these, non-febrile equality was observed from evening to morning in one case of chronic phthisis. Among the "low temperatures" it was also observed from evening to morning in one case, and from morning to evening in two cases. With these exceptions, in which it was frequent in these individual cases, such equality is rare among the whole number of observations. In three cases, however, equality of temperature from evening to morning was met with respectively in 37·5, 33·3, and 33·3, per cent., and in two more in 15 and 16 per cent. of the observations; in all the rest it did not occur in more than 10 per cent., and in a considerable number in both categories it was limited to a single observation. It, however, also occurred from morning to evening in one case in 42·5, in one case in 20, and in eight cases in from 10 to 20 per cent. of the observations. In the remainder it was below 10 per cent.

Class.	No. of cases.	Morning equal to preceding evening.	Morning equal to following evening.
Acute tuberculosis	8	4	3
Acute phthisis..............	24	9	14
High temperatures.........	8	5	5
Chronic phthisis............	15	9	10
Low temperatures	20	9	11
Total.................	75	36	33

In some cases, where the morning temperature has been identical with that of the preceding evening, there is a fall, but in others a rise is observed to that of the following evening, or, as in one case observed, there may be a gradual fall continuing through thirty-six hours, but followed by a fresh exacerbation. This approximate equality of temperatures in the mornings and evenings is, however, commonly associated with high pyrexia and with a severe form of the disease.

§ V. *Remissions and exacerbations.*—The remissions from evening to morning, and the exacerbations from morning to evening, correspond pretty closely, at least in the higher ranges (see Tables 7 and 8). Marked remissions and exacerbations do not, however, necessarily follow one another, as a continuous or quasi-continuous temperature appears in some cases to be maintained, though generally only during short periods. Extremes of either correspond usually to an alternation of a high and a subnormal temperature. The maxima recorded in these cases are a remission of 8·8°, and an exacerbation of 8·4°, which immediately succeeded one another, and were also followed by a series of considerable though not equally great alternations, occurring during six days preceding death.* Two out of 75 cases had

* The temperature in this case at this period were—

Evening 103·8°.	Morning 95°.
„ 103·4°.	„ 95·2°.
„ 102·2°.	„ 95°.
„ 100°.	„ 98°.
„ 102°.	„ 96°.
„ 99·2°.	„ 97·4°.

Death.

The maximum morning temperature in this case was 102·2°, the maximum evening 103·8°; but the average of the morning temperatures was 98·6°, of the evening 100·7° (25 days). The average of the remissions from morning to evening was 3·9°, of the exacerbations from morning to evening 3·3°. Remissions from morning to evening also occurred to the extent of 4·2°, and exacerbations from evening to morning of 4·8°. The morning temperature was higher both than the preceding and also than the subsequent evening respectively in 25 per cent. of the observations. The morning temperature was normal or subnormal in 37·8 per cent. of the observations. The evening

remissions of from 6° to 7° from evening to morning, and both these cases also had exacerbations of the same extent from morning to evening.* Five† had remissions of from 5° to 6°, but in none of these, however, did the exacerbations reach the same height, though attaining to 4°. Two, however, had exacerbations of from 6° to 7° and from 5° to 6° respectively, without a corresponding degree of remissions.‡ Eight cases, therefore, out of a total of

was normal after a morning pyrexia in 1 observation.—Acute pneumonic phthisis of nine months' duration; much sweating; much diarrhœa. The patient was a drinker. He had worked at Woolwich, and there was a suspicion whether he might have been subjected to malarial influences; but there was no history of this, nor enlargement of the spleen. Quinine taken regularly had no effect on the temperature. M. Lebert records a case (loc. cit., p. 63) where acute tuberculosis supervened after intermitting fever; and the types of the pyrexia observed in the two diseases were markedly different.

* One case of chronic phthisis with an acute exacerbation and recent miliary tubercles. These remissions were on two occasions from 101·4° to 94·8°. They were followed by three days of subnormal temperature preceding death. In another case of acute phthisis a fall was observed from 104·4° to 97·8°, and another from 103° to 98°. These were both followed by nearly corresponding exacerbations.

† One of these was among the cases of acute tuberculosis, 1 in acute phthisis, 1 in a case of chronic phthisis with acute exacerbations, 1 among the "high temperatures," and 1 among the "low temperatures." In the two last named the fall might be regarded as almost critical. Thus, in the case with high temperature the fall was from evening 103·4° to 98°. After this the morning temperatures never exceeded 98° during twelve days; and the evening temperatures fell nearly a degree for four successive evenings to 99°, and remained at 99° for two evenings, and then fell in five evenings to normal, and the patient was dismissed convalescent. The cough and expectoration and the sweating, all of which had been severe on admission, had then entirely ceased. There were consolidation and other signs of phthisis at both apices. In the case occurring in the class of "low temperatures" the remission occurred on the morning after admission (evening 101·4° morning 96·2°). There was no remission of the same extent after. The temperature gradually fell to normal. This case was marked by much greater disturbance.

‡ Both cases of acute phthisis. One exacerbation from morning 100° to evening 106·6°, the only occasion on which so high a temperature was reached in the case. The greatest remission succeeding this was to 102·4° (4·2°). In the other, though the greatest exacerbation was from [illegible] test remission was from 3° to 4°,

75, or 10 per cent., exhibited in their course remissions exceeding 5°; and five, or 6·6 per cent., had exacerbations of a corresponding extent.

The larger remissions and exacerbations, with the exception of those just alluded to, are, as a rule, nearly equally distributed between the fatal cases of acute tuberculosis, acute and chronic phthisis, and the cases of high temperature. The class of low temperatures not ending fatally shows these, however, to a much smaller extent.

This may be seen from the accompanying table constructed from Table 9. Each column shows the percentage of all remissions and exacerbations exceeding the degree specified.

Class of cases.	REMISSIONS. *Evening to morning.* Percent. of all remissions observed in every case in each class.				EXACERBATIONS. *Morning to evening.* Percent. of all exacerbations observed in every case in each class.			
	More than 3°.	More than 2°.	More than 1°.	Less than 1°.	More than 3°.	More than 2°.	More than 1°.	Less than 1°.
Acute tuberculosis	16·6	44·4	69·5	30·5	20·9	34·6	65·1	34·9
Acute phthisis	12·3	29·7	60·2	39·8	9·2	23·2	49·8	50·2
"High temperatures"	11·2	27·4	64·3	35·7	15·4	34·3	60·9	39·1
Chronic phthisis	11·8	33·6	62·4	37·6	4·7	19·	59·2	40·8
"Low temperatures"	2·7	10·4	36·5	63·5	2·	8·	37·2	62·8

It will be observed in comparing the columns of exacerbations with those of remissions, that in the class of acute phthisis there is less approximative correspondence between them than is observed in the other classes. This appears to be due in part to a greater tendency to continuity of temperature, in part to the more frequent occurrence of remissions from morning to evening, in this than in other classes. It is also in part accidental, owing to

the inclusion in it of two cases where the remissions and exacerbations were throughout small, though they ran an acute course.* A table almost identical with the preceding might be constructed out of the numbers of cases, showing the average of all the remissions and exacerbations occurring in each case. (See Tables 7 and 8.) It is only in the class of "low temperatures" that these averages fall, in any large proportion of the cases, below 1°.

Professor Niemeyer† has asserted that tuberculous phthisis may be distinguished from the pneumonic forms by the larger remissions prevailing in the latter, and that when tuberculosis supervenes these remissions disappear or diminish. This appears, as far as the evidence of the cases here analysed is concerned, to be completely disproved by their results; for a greater proportion of large remissions and exacerbations are observed in acute tuberculosis than in any other class, though the difference is not very considerable. If to these we add the class of cases where acute tuberculosis is attended by a low temperature, we may conclude with Wünderlich that from a thermometric point of view there is no feature which can be relied on as a characteristic to distinguish these two forms.‡ The only point

* In one of these the temperature was low, the maximum morning not exceeding 101·3°. In the [illegible] remissions [illegible] evening and the exacerbations from [illegible] verse (maximum [illegible] remission morning [illegible] morning to [illegible] morning 1·6 [illegible] evening on [illegible] days. Alm[illegible] the temp[illegible]

† 'Kün[illegible]

‡ 'Die [illegible] insisted [illegible] forms of [illegible] that cases of [illegible] very diff[illegible] in the m[illegible] also have [illegible]

which, with occasional exceptions, may be affirmed of the different classes is, that the higher the fever, the greater is the liability to marked remissions and exacerbations; but, on the other hand, a few cases with high fever show an approximative uniformity of temperature.

Although, as before shown, in a large number there are remissions from morning to evening, and exacerbations from evening to morning, yet these in the majority are less in extent than the remissions from evening to morning, and the exacerbations from morning to evening. (See Tables 7 and 8.) Thus, in 75 cases, only 1 case showed a remission from morning to evening exceeding 4°, and only 4 cases an exacerbation from evening to morning of this extent. In a large number also, these latter were limited to one or two observations, and the majority of the remissions and exacerbations in these did not exceed 0·5°, and sometimes ranged as low as 0·4°, 0·2°, or even 0·1°; so that in such cases there was an approximative though not an absolute attainment of what may be regarded as a nearly equal temperature between the mornings and evenings.*

There are, however, a certain number of cases where high morning temperatures predominate, in which the major remissions occur from morning to evening, and the major exacerbations from evening to morning; and these may be sufficiently numerous throughout the case to constitute higher averages for these remissions and exacerbations, than for those occurring in the more usual order; and these

M. Lebert has observed, exceptional. He remarks also that the same variations may be observed in tubercular peritonitis and meningitis (l. c., pp. 58, 61, 78-9).

* The number of the smaller remissions from morning to evening (below 0·5°) is pretty equally distributed among the different classes, with the exception of acute tuberculosis, which shows a less proportion. There is throughout, also, a nearly equal proportion of the minor exacerbations from [illegible] to morning, though these are more numerous in acute tuberculosis [illegible] in the rest. In all, with the exception of the class of acute tuberculosis, [illegible] number of the minor exacerbations than of the minor [illegible] differences in the class of acute tuberculosis is

cases form no inconsiderable proportion of the whole number analysed. This may be seen by the accompanying table, which gives only the number of cases in each class in which remissions from morning to evening, and exacerbations from evening to morning, were observed.

Class of cases	Remissions					Exacerbations				
	No. of cases.	Max. M. to E. greater than E. to M.	Max. M. to E. = E. to M.	Av. M. to E. greater than E. to M.	Av. M. to E. = E. to M.	No. of cases.	Max. E. to M. greater than M. to E.	Max. E. to M. = M. to E.	Av. E. to M. greater than M. to E.	Av. E. to M. = M. to E.
Acute tuberculosis ..	5	0	0	0	0	[illegible]	0	2	0	2
Acute phthisis	22	6	4	4	3	21	5	4	3	3
"High temperatures"	8	2	0	2	2	8	1	1	1	3
Chronic phthisis	15	2	0	0	3	15	0	5	1	4
"Low temperatures" ...	19	10	0	7	5	18	5	0	6	0
Total	69	20	4	13	13	68	11	12	11	12

Although, however, the maxima of these remissions and excerbations may be respectively higher than the converse, yet the averages of all such occurring in the case may be equal to or less than those of the remissions from evening to morning, and of the exacerbations from morning to evening.*

Other variations are occasionally found. Thus, in one case, though the remissions occurring from morning to evening were greater than those from evening to morning,

* Thus, in one case the maximum and average morning temperature was higher than the maximum and average evening temperature; yet the remissions from evening to morning were greater than those from morning to

yet the exacerbations from morning to evening when these occurred were also greater than those from evening to morning.

The alternation of these remissions and exacerbations with those following the more regular course produces an irregularity in the latter, which disturbs the order of the temperatures in a large number of cases of phthisis. This has been already alluded to, but the final effect is to produce a want of correspondence between the remissions and exacerbations of either class, which is illustrated in Table 10; where it is seen that in a considerable proportion of cases their is no equality observed between them.

§ VI. *The cause of the variations of temperature* now described is, in the majority of cases, by no means distinct. In more than one case the highest temperature observed was that on the evening of admission, showing that fatigue and excitement may increase the fever. I have more commonly than the reverse failed to find any positive evidence of increase of the physical signs following severe excerbations. In some instances, however, they preceded distinct and fresh pneumonic consolidations. Rapid and extensive softening also sometimes produces this effect, and in some cases they indicate other intercurrent inflammations.* This has been observed in a suddenly developed high morning temperature, on the supervention of parotitis, by Jochmann;† but in a similar case in my own experience, no marked elevation of temperature was observed.‡ Rigors sometimes attend these exacer-

evening, and the exacerbations from morning to evening were greater than those from evening to morning.

M. Max. 103°, Av. 101°8. E. Max. 103°, Av. 101·2°.

Max. Rem. E. to M. 4·9°, Av. 1·6°. Max. Rem. M. to E. 3·2°, Av. 1·2°.

Max. Exac. M. to E. 4·5°, Av. 1·8°. Max. Exac. E. to M. 3°, Av. 1·4°.

[illegible] morning temperature was, however, higher than the previous evening [illegible] cent., and higher than the following evening in 65 per cent. of 18 [illegible]

[illegible] carditis," Wun[illegible]erlich, 'Arch. der Heilk,' 1860, Case ii.

[illegible]

[illegible] fore death, but only one evening tem- [illegible] 99·8°.

bations, but in a distinct form they are in my experience not very frequently observed. In one case, where rigors were of repeated recurrence, with great exacerbations of temperature (106·1°), the invasion of pneumonia was observed successively at both bases.* Rigors were common in Louis' observations with the evening exacerbations of the fever, being wanting only in 16 out of 95 cases; they were rare at other times. My own enquiries have not been very specially directed to this point; but they have not appeared to me to be complained of with this frequency.†

Marked exacerbations are commonly followed by equally great remissions. The latter, as Jochmann has observed, signify in all probability a condition of "inanition," "exhaustion," or "collapse."‡ The temperature in these remissions may sink as low as 95°, but it rarely falls below 96°. M. Lebert has, however, known it fall to 90·5° F. In some cases, however, they form a sort of pseudo-crisis, or (as in two instances) they may constitute a real crisis similar to that observed in acute pneumonia, the temperature scarcely rising subsequently, and within a short period regaining the normal standard. In some instances M. Lebert has observed a subnormal temperature maintained for weeks

* In this case the fever was occasionally quotidian, but was also tertian for six days, but the rigors returned at irregular hours. Thus:—October 17th, rigor at 1.30 a.m.; 18th, rigor at 10.30 p.m.; no fever for twenty-four hours before; 19th and 20th, apyretic until 5 p.m. on the 20th; 21st, apyretic; 22nd, rigor at 1 p.m.; 23rd, slight pyrexia, rigor not noted; 24th, rigor at 10 a.m.; 25th, no notes (probably no rigor); 26th, rigor 6·45 a.m.; 27th, pyrexia, no rigor; 28th, pyrexia in morning; 29th, no notes; 30th, no pyrexia, morning or evening; 31st, rigor; November 2nd, rigor. On those days when the temperature was taken at frequent intervals, the pyrexia lasted in one twenty hours, in one twelve hours, and in one fourteen hours. It was not observed to fall below 98°. On two occasions it reached 106°, on one 105·4°, and on another 104·6°. The patient became discontented and left the hospital.

† Andral, 'Clin. Med.,' vol. iv, p. 174, says that rigors are rare in the evening.

‡ Jochmann quotes Chossat's observations to show that during inanition the oscillations of temperature are increased.

after the subsidence of the pyrexia, ranging from 97·7° to 96·8° or even to 95·9°, with general improvement of the health and gain of weight.

A marked and increasing intensity of remission with high exacerbations may, in some acute cases, for several days precede death. Great remissions, therefore, without a corresponding diminution of the exacerbations, cannot be regarded as a favorable sign. Other conditions of exhaustion may in some cases produce an apyretic or even a subnormal temperature lasting for some days. One such was illustrated* by a female patient who miscarried while in the hospital, another by the supervention of perforation of the pleura,† though this event is not always followed by such a result as was observed in another and similar case, though here also a fall to normal on the next morning followed this event.‡

A subnormal standard may also be attained for some days before death, but this is by no means invariable; and

* A chronic case admitted with a recent fresh attack. Temperature during forty-three days scarcely ever exceedod 100°. A miscarriage was followed the next morning by a temperature of 95·2°, next morning 98·4°, evening 100·2°, next morning 102°. Continued higher in the mornings than evenings for three days, morning 102°, evening 101°. Then for twenty-one days evening higher than morning with great remissions, but never intermittent, remissions being generally 3°, and once 5°, followed by an exacerbation of 5°; two such exacerbations occurring. The last was attended by the invasion of acute pneumonia, which proved fatal.

† The fall of the temperature did not *immediately* follow the perforation. It had been high before—103·8°, on the morning on which it occurred; on the evening of the day after the perforation it was 103·4°, and the next evening 104·4°; then ensued a gradual fall during four days, until on the seventh day after the perforation the temperatures were morning 97°, evening 96·8°. On the eighth day, morning 99·4°, evening 99·6°; after this a rise took place, but the temperature continued on the whole low, but never again subnormal during forty-four days until death.

‡ M. Lebert, l. c., p. 81, has found in some cases the invasion of pneumothorax associated with a *rise* of temperature, followed by a considerable fall, and finally returning to the ordinary type of the pyrexia. If the opening closes the temperature may gradually fall. If pneumo-thorax occurs early in the disease it may be followed by an arrest of this and a gradual subsidence of the pyrexia.

in many cases, the temperature continues high up to the fatal termination.

Suffocative bronchitis attending tuberculization may also be attended by a generally low, and by subnormal temperatures; and this may possibly explain some cases where these are observed in acute tuberculosis; but intense dyspnœa, with cyanosis, may be observed when the temperature is 102° or 103°. In some cases also in all forms, as already remarked, normal temperatures may be abruptly interposed in the midst of a general pyrexial course, and this without any evident cause; though Jochmann is disposed to attribute these, and also periods of lower temperature intervening after this had been previously high, to the effects of exhaustion, while in his opinion sustained high temperature may be an indication of maintenance or increase of the vital power.*

Lardaceous disease of the liver, spleen, kidneys, or other viscera, among the chronic cases is not unfrequently associated with an average low temperature; but it does not determine this, for it is also met with among highly pyrexial cases, both in the chronic and acute varieties.†

Diarrhœa may also be attended with a tendency to great remissions; but what part it may play in their production, independently of severe exacerbations, has yet to be more fully studied. It is, however, certainly one of the most important factors conducing to exhaustion, and it also appears in many cases to be the most prominent feature in cases where large remissions are frequent or repeated. In

* Loc. cit., pp. 64, 66.

† Thus, I have observations of the temperature in 12 cases in which lardaceous disease was found; 7 of these were chronic cases, and 5 acute. Of the chronic cases the maximum temperature exceeded 103° in 5, and 102° in 2. The average temperatures (either of the morning or evening) exceeded 102° in 3 cases, 101° in 1 case, 100° in 2 cases, and 99° in 1 case. Of the 5 acute cases the maximum temperature exceeded 101° in 1 case, 103° in 3 cases, and 104° in 1 case. The average temperatures (morning or evening) exceeded 104° in 1 case, 103° in 1 case, 102° in 1 case, 101° in 1 case, and 99° in 1 case. M. Lebert remarks that lardaceous diseases has but little effect on the temperature.

some cases, however, as remarked by M. Lebert, diarrhœa and ulceration of the intestines have but little effect on the fever.

The conclusion can hardly be avoided that the larger oscillations may be in part due to the peculiar type of the fever. In this it resembles the pyæmic fever of blood infection* or of internal suppuration; and as these characteristics chiefly occur in acute cases or in the intensifications of chronic cases, some part of the phenomenon may be due to constitutional conditions, and particularly to secondary invasions of other organs.† In what respect the temperature *per se* is attributable to the tuberculization must yet be a subject for further inquiry. Dr. Ringer was disposed to regard the severity of the fever as a measure of the intensity of this process, but some cases of acute tuberculosis appear to form exceptions to this position. Very high temperatures may indeed be met with when there are few other elements of inflammation present, and acute tuberculosis certainly presents some of the highest ranges of pyrexia found, though this is occasionally equalled or nearly equalled by some of the pneumonic forms of the disease.

In the other varieties of phthisis coming under my observation, the intensity of the pyrexia has generally appeared to be largely proportioned to the extent of the intercurrent inflammation. Lebert, however, has remarked that this is not invariably the case, and that high fever may exist with little local mischief, or without breaking down of the lungs, while moderate fever may be found where the lesions are extensive and the pulmonary destruction is considerable; and I am disposed to believe with him, that these differences depend either on the constitutional‡ pecu-

* A remark for which I am indebted to my friend and colleague Dr. Burdon Sanderson. Andral, 'Clin. Med.,' iv, p. 235, pointed out the resemblance between the sweating in phthisis and that occurring in suppurative fever. He attributed the sweating to the softening of tubercles.

† These latter, however, do not always determine pyrexia.

‡ Lebert remarks that the *age* of the patients affected has little influence [on the se]verity of the pyrexia.

liarity of individual patients, or on varieties in the pyrogenic effects of the changes in the lungs which require further elucidation.

In the main, however, the different types defined by Jochmann correspond very closely with the different varieties of the disease, though his observations do not include cases of acute tuberculosis. The slighter forms of fever (Type I, and Variety A, Type II) correspond either with the early stages of the disease, with mild exacerbations, or with very slowly progressive disorder associated with fibroid thickening or with little suppurative action; though the latter when present, as shown by not inconsiderable expectoration, does not necessarily determine high pyrexia. The other varieties are associated either with recent pneumonic invasion and considerable extension of the tuberculizing process, with or without rapidly progressing ulcerative action. But it must be again remarked, that no special type corresponds with any individual varieties in the anatomical forms of the disease.*

Perspiration cannot *per se* be held to cause remissions of the temperature; it appears in the majority of cases to be a consequence and not a cause of its fall, though judging from analogy, it may possibly mitigate the severity of the pyrexia. It is most marked, as a rule, in the strongly remitting forms, and it bears in this respect a strong analogy to what is observed in other forms of hectic fever, and particularly in purulent infection. It may, moreover, be observed when the temperature is high; and the temperature may even appear to rise while it is present.† In some

* Lebert states that "perialveolitis," "peribronchitis," and "periarteritis," show the same variations of temperature as large cavities proceeding from "broncho-alveolar" inflammation.

† Thus, in a chronic case, where the temperature was on the whole low (average morning 99°, average evening 99°), and where during sixteen days it had not exceeded 99°, and on four occasions was normal, a rigor ensued at 6.30 a.m. on the seventeenth day. Temperature, twenty minutes after the rigor, 101·2°; fell in one hour to 100·4°, when sensation of heat was felt by patient. Two hours later sweating, during which the temperature rose to 101·4°. In the next hour it had fallen to 99°, and in the ensuing two hours

exceptional cases, also, of which I have seen an instance, the remissions may be unattended by this symptom. In some of the cases when the temperature fell from the morning to the evening, profuse sweating occurred during the day. Jochmann, however, considers that in some cases it may be critical, and may alternate with other discharges.*

Hæmoptysis, as I have more than once observed, even when the amount of this has been considerable, does not appear to exert any influence on reduction of temperature. I have known the temperature rise on its occurrence, and have also known the temperature fall before the hæmoptysis had completely ceased. In some cases, no fever follows a first attack of hæmoptysis, and very high pyrexia may be observed after a second attack.

Pleurisy complicating phthisis may be associated, but not constantly, with a rise of temperature. When it occurs in the earlier stages it may determine a distinct invasion of pyrexia. Lebert remarks that in these cases tapping is usually followed by a fall of temperature, but that if the fluid become fetid, increased pyrexia ensues, which, however, may be again diminished by antiseptic injections. The subsequent course of the fever is determined by the progress of the tuberculosis. I can confirm by my own experience the result of M. Lebert's observations on the effect of paracentesis.

Perforation of the intestines has been observed by Lebert to be followed by a fall of temperature amounting to collapse.

Phthisis occuring in the puerperal state is usually one of the most acute and fatal forms of the disease, and the temperature generally ranges high ; but all the variations previously noticed may be observed. Most commonly the

to normal. It continued normal throughout the day, and on the following day had resumed its previous type.

* Loc. cit., p. 80. A case where diarrhœa supervened with the fall of temperature. On the cessation of this, profuse sweating appeared under the same conditions. Jochmanu, therefore, thinks it undesirable to check critical sweating. The chief question is, however, in what sense the term "critical" is used.

remissions are great; less frequently a continuous high pyrexia is observed, or it may rise gradually. In a few cases a gradual improvement is noticed with diminution or arrest of the pulmonary disease.*

The sense of heat experienced by the patient, as observed by Jochmann, does not necessarily bear any direct relation to the temperature. Rigors, as is well known in other diseases, are almost invariably at the time of their appearance already attended by an elevation; and in other cases the extremities may be cold when the temperature of the trunk is actually febrile.

In the earlier stages even of comparatively acute attacks a high temperature is not necessarily indicative of a continuously progressive course; for the temperature in such cases often tends to fall, in accordance with what has been already stated to be observed in some of these cases, viz., that they not unfrequently pass into subacute or chronic types.

Even in patients coming under observation with recent exacerbations, time alone can in some cases determine in what the future event of such attacks may eventuate; and in a fair proportion, a speedy cessation of the fever and also of the more threatening symptoms may be reasonably looked for. Thus, out of 21 cases, presenting on the whole low temperatures, there was a nearly absolute loss of pyrexia in 5, a complete disappearance in 3; total 8. In those where the disappearance of pyrexia was not absolute there was only an occasional exacerbation to 99°. In 8 cases with high temperatures there was absolute cessation in 2, a nearly absolute in 2, total 4; or out of 29 cases not ending fatally there was an almost complete or a complete disappearance of fever in 12 or in 41·3 per cent. The reduction of temperature followed in several of the cases at very short periods after admission to hospital. In 3 cases (2 of high temperatures and 1 of low), the patient was admitted after a recent attack commencing with rigors and pains

* See Lebert, loc. cit.

in the limbs; and the temperature in these fell respectively to normal within the twelfth, fifteenth, and nineteenth days after the attack. In all these there was evidence, in addition to consolidation of the apices, of recent affections of the bases. In the whole number, the fall of temperature occurred in the majority, comparatively soon after admission; in two within less than a week, in 4 within less than two weeks, in 3 more within three weeks. In 3 only was the stay in hospital protracted to four weeks, ten weeks, and thirteen weeks respectively, before a reduction in temperature ensued. In 4 only were the patients dismissed in less than a week after the reduction; in the others a normal temperature, or one only broken by slight exacerbations not exceeding 99°, was maintained for periods varying from twelve to fifteen days prior to their discharge.

In all these cases the most noticeable improvement was in the disappearance of the râles. In 3 cases minute notes were not taken on the patient's discharge; but in 9, their complete or almost total disappearance was observed in 7, or 77·7 per cent. In 7 out of these 9, there were more or less distinct signs of cavities at one or both apices; in the others, there was consolidation with râles. In 6 also out of the 9 there was consolidation at the bases. In none was there observed any diminution of the dulness at the apices, but in 2 the dulness had left the bases, coincidently with the fever. This was especially remarkable in one case admitted, four days after a rigor, with a temperature of 103·6°, when after nineteen days the temperature fell to normal, and the dulness and râles at the bases disappeared.

Complete disappearance of the cough was rare, being only noticed in one case; but in 6 out of the 12 a great diminution of expectoration was observed. Two cases were admitted within a few days after attacks of hæmoptysis. In one of these, however, the pyrexia ceased eleven days after admission, while the hæmoptysis continued for five days longer.

Improvement in the digestive functions was also a marked feature. In 7 cases where there was a furred tongue, nausea or vomiting, and loss of appetite, these symptoms

had improved or disappeared in all. In one only was no improvement noted. In 4 cases where diarrhœa existed, this also ceased.

The weights were, unfortunately, not accurately taken in these cases. In one there was a gain of 4 lbs. in thirty-two days, during sixteen of which the temperature was above the normal; and the greatest gain was during the first period, being 3 out of the 4 lbs. In one case, where œdema of the vocal chords and epiglottis, hoarseness, and loss of voice existed (together with hæmoptysis) on admission, these symptoms disappeared after the application of nitrate of silver to the larynx.

The characters of the pyrexia in these cases presented great variations, and no single feature common to all. Among those with low temperatures, one, a female, already alluded to, had throughout a morning temperature higher than the evening. One other of these had a maximum temperature exceeding 103°, and another exceeding 102° but the average of these did not exceed 100° for evening or morning. The remissions and exacerbations were moderate; in one case only exceeding 3° for the former, and in two only for the latter. In one, however, in addition to that already alluded to, the morning temperatures were always higher than those of the subsequent evening, and in 40 per cent. of the observations they were higher than those of the previous evening. In 3 others the morning temperatures were frequently the highest in 21, 28, and 40 per cent. of the observations respectively, and with nearly the same frequency as compared with those of the subsequent evening.

The cases with high temperatures presented no peculiarities as a class, but the higher morning temperatures were not so frequently observed as in other cases where no reduction occurred. In all of them the maximum temperature exceeded 103°, and in one case 104°; the average evening temperatures being in three cases above 100°, and in one 101·2°. The remissions from evening to morning were never less than 2°, and in one case 5·4°. The exacer-

bations from morning to evening were 1·4° in one case, above 2° in two cases, and 4·4° in one case.*

In all these cases, both of "low" and "high" temperatures, the treatment was practically indifferent. Small doses of quinine and iron were given in a few, and the rest of the treatment was directed to special symptoms. Gallic acid or ergot for hæmoptysis; bismuth, soda and calumba for stomach complications; opium and astringents for diarrhœa; and morphia, senega and counter-irritation for cough. Cod-liver oil was given in nearly all. The conclusion, therefore, appears to be that in a fair proportion of cases, some of which present symptoms of considerable severity, a reduction or cessation of pyrexia and improvement or arrest of active lung-changes, follows within certain periods, and in some instances within very short periods after confinement to bed and good hygienic management.

Observations on the Pulse.

The circulation is accelerated, and usually in proportion to the intensity of the disease.†

In the severer cases the pulse is rapid, whether the

* Lebert, l. c., p. 71, who has also observed this tendency to natural subsidence of the fever in some cases, records one in which a slow fall of temperature extended over nearly four months. November, morning 100° to 102°, evening 103° to 104°; December, morning 100°, evening 102°; January, morning nearly normal, evening 99·5° too 100·8°; February, occasional exacerbations in the evening; March, morning normal or subnormal, evening occasionally 100°.

† See Tables 11 and 12. These and the following tables are formed from observations on the temperature, pulse, and respiration, taken night and morning. The means are only calculated when the number of observations is sufficient to afford a fair average; hence they represent a smaller number than the preceding tables. Those cases only have been taken which give the data in all these points. It must further be borne in mind that these tables are formed from severe cases of phthisis, of which a large proportion proved fatal. The data are, therefore, scarcely applicable to chronic forms, or to the intermediate periods, when but little progress is observed.

disease be in the acute or chronic form, and acceleration is almost invariably observed whenever the disease is progressing. Thus, pulses above 140, both in the morning and evening, were observed, not only in the acute and chronic forms, but also in some of the non-fatal cases, both with "high" and "low" temperatures. The acceleration is not, however, constant; and in all forms (even including cases of acute tuberculosis without cerebral complication) the pulse may be less than 100, not only occasionally, but with a frequency sufficient to reduce the mean of a series of daily observations below this rate, and it may also in fatal cases occasionally fall to less than 70 both in the morning and evening.

The slower pulses are, however, found in the greatest numbers in the non-fatal cases, and especially in those presenting "low" temperatures, where a large proportion of the mean pulses will be found below 90, 80, or even 70, and a very considerable proportion even of the quickest pulses are below 100. In all the other forms, however, pulses exceeding 100 are almost invariably observed during the progress of the case.

Dr. Edward Smith, in a valuable paper on the rate of respiration and pulsation in phthisis,* found that the rate of the latter fell after 9 p.m. and rose in the morning. Comparing, however, the morning and evening observations (Table 12), it will be seen that, of the quickest pulses, above 100, above 120, and above 140, a large proportion is met with in the evening; while of the slowest pulses, in the means above 100, a similar excess obtains for the evening. The difference, however, varies in each class of cases, and in a certain proportion equality prevails between the maxima and minima, and also between the means of the morning and evening pulses.

* 'Med.-Chir. Trans.,' vol. xxxix. Dr. Smith found the pulse to fall after 9 p.m., and to rise after 5 a.m. The observations here analysed were made from 9 to 11 a.m., and from 8 to 10 p.m. Dr. Smith's cases were observed throughout the twenty-four hours. These only refer to the clinical experience of morning and evening visits.

The relation between the morning and evening pulses is shown in the annexed table, where it is seen that of the quickest pulses observed, the larger number occur in the morning, which so far confirms Dr. E. Smith's observation. In the slowest and mean pulses, however, the reverse obtains.

	Quickest pulses. 54 cases.	Slowest pulses. 54 cases.	Mean pulses. 46 cases.
Evening pulse quicker than morning............	20 cases	33 cases	28 cases
Evening pulse slower than morning............	22 cases	14 cases	12 cases
Evening pulse equal to morning	12 cases	7 cases	6 cases

The discrepancy between this table and the columns relating to the quickest pulses in Table 12 is explained by the fact that the percentage of the morning pulses is calculated on 74 cases, and that of the evening on 54.

Observations on Respiration.

The respiration is also accelerated, generally, also, in proportion to the intensity of the disease; but even in the non-fatal cases a considerable number present a mean both of morning and evening respirations above 30 to the minute (see Tables 14 and 15).*

It will be seen (Table 15) that there is an excess of the

* The means thus found are higher than those given by Dr. E. Smith (l. c.). These means are those of all the respirations in each case, for morning and evening. An absolute table of the quickest and slowest respirations observed has not been constructed. The tables given are of respirations corresponding to the quickest and slowest pulses; but, though not always corresponding, they present a close approximative estimate of the characters of the respirations.

means of respirations above 30 in the evening, as compared with the morning, in the proportion of 65·2 to 56·7 per cent.; and also that there is a slight excess in the evening above 20.

	Resp. accompanying quickest pulse.		Resp. accompanying slowest pulse.		Mean respiration.	
	M. Per cent. of cases.	E. Per cent. of cases.	M. Per cent. of cases.	E. Per cent. of cases.	M. Per cent. of cases.	E. Per cent. of cases.
Respiration above 20	98·6	100	93·2	96·2	95·9	97·8

When, however, the respirations in the morning and evening are compared in individual cases, it will be seen that in a fair proportion they are equal in frequency, and that those where the evening is in excess are below half of the whole number.

	Respiration of quickest pulse. 54 cases.	Respiration of slowest pulse. 54 cases.	Mean respiration 46 cases.
Evening respiration quicker than morning	25 cases	24 cases	20 cases
Evening respiration slower than morning	23 cases	17 cases	20 cases
Evening respiration equal to morning	6 cases	13 cases	6 cases

On the Pulse and Respiration in relation to the Temperature.

When the pulse [illegible] in relation to the [illegible] other, the

following facts appear to result from a further analysis of these tables :

(*a*) *Relation of pulse to temperature.*

There is no necessary relation between the pulse and temperature; a very rapid pulse may be attended with a low temperature both in the morning and evening, as—

Morning pulse, 140.	Temperature, 97°	} 2 cases.
„ „ 120—130.	„ 99°—100°	} 2 cases.
Evening pulse, 130.	„ 95°— 96°.	

And, even of the mean pulses, these may exceed 130 during five days before death with a mean temperature of 100°.*

A high temperature may also be associated with a comparatively slow pulse, and even in acute tuberculosis the same disproportion between the pulse and temperature may be occasionally observed, particularly when those of the morning and evening are contrasted.†

Very different temperatures may even in the same case be also associated on different occasions with a pulse of identical frequency. In one case temperatures in the morning of 104° and 100·2° were respectively found on different days with a pulse of 108.‡

Although, therefore, no constant ratio can be established between the pulse and the temperature, yet cases distinguished by rapidity of pulse are most commonly those where the temperature is the highest, and, conversely, uniformly low pulses are generally those where low temperatures prevail. Thus (see Tables 11 and 13), of the

* A chronic case, dying five days after admission to the hospital.

† Case.

Quickest pulse,	morning = 128.	Temperature	100·8°.
„	evening = 120.	„	102·8°.
Slowest pulse,	morning = 82.	„	101·4°.
„	evening = 78.	„	105·2°.
Mean pulse,	morning = 94.	„	101·5°.
„	evening = 100.	„	102·8°.

‡ ...man, l. c., observed that a pulse of the same frequency might exist ... temperatures of mid-day and evening as with the lower tempera-...g.

cases where the quickest pulse did not exceed 90, all but one are in the class of "low" temperatures. This association, however, is more marked in the morning than in the evening. Thus, of nine cases where the quickest pulse in the morning did not exceed 100, in two only was the morning temperature higher than 100°, but of six similar cases in the evening, in two only was the temperature below 100°.

Even the slowest pulses observed in individual cases have a tendency to be associated with lower temperatures, and in the mean pulses the same relation may be observed. Thus—

Slowest morning pulse below 100 = 57 cases. Temperature below 100° in 41 cases, or 71·9 per cent.

Slowest evening pulse below 100 = 34 cases. Temperature below 100° in 19 cases, or 55·8 per cent.

Slowest morning pulse below 80 = 22 cases. Temperature below 100° in 18 cases, or 81·8 per cent.

Slowest evening pulse below 80 = 13 cases. Temperature below 100° in 9 cases, or 69·2 per cent.

Mean morning pulse below 100 = 32 cases. Temperature below 100° in 24 cases, or 75 per cent.

Mean evening pulse below 100 = 16 cases. Temperature below 100° in 8 cases, or 50 per cent.

Mean morning pulse below 80 = 8 cases. Temperature below 100° in 8 cases, or 100 per cent.

Mean evening pulse below 80 = 4 cases. Temperature below 100° in 4 cases, or 100 per cent.

(The absolute numbers of morning and evening pulses, as here stated, bear no relation to one another, since the evening pulses are de[illegible] from [illegible] of cases.

That the accelerat[illegible] direct relation to th[illegible] when the evening [illegible] compared with th[illegible] perature is observ[illegible] of cases th[illegible]

Thus, in [illegible] in 46 cases [illegible] observed :—

	Quickest pulse. Per cent. of cases.	Slowest pulse. Per cent. of cases.	Mean pulse compared with mean temp. Per cent. of cases.
Evening temperature, highest ...	74	75·9	76·0
Evening pulse, quickest	87	61·1	60·8

When, however, the evening pulse was slower than the morning, the evening temperature was higher in 59 per cent. of such cases among the quickest pulses, in 64 per cent. among the slowest pulses, and in 58 per cent. among the mean pulses (in the latter the comparison is between the means of the morning and evening temperatures). (See also p. 435 and Table 22, Appendix.)

(*b*) *The relation of the respiration to the temperature* appears, as far as these cases show, in respect of given rates of frequency as compared with degrees of heat, to be almost indifferent, except that in the evenings when a low temperature (*i. e.*, below 100°) is found, there is a rather larger proportion of cases of moderately slow respiration; and the most rapid breathing in the evening is associated with temperatures above 100°. In the morning, however, these relations are exceedingly irregular.* It may, however, nevertheless be occasionally observed in individual cases that there is a very close correspondence between the rises and falls of temperature and increase and diminution in the frequency of the respiration.†

(*c*) *The relation of the respiration to the pulse* is more definite than it is to the temperature, though here also great variations may be observed. Quick breathing

* This statement is founded on an analysis of Table 16. As the results ... almost negative, it appears unnecessary to give the details, especially as ... involve complex figures. The highest observed morning temperatures, ... 106° (2 cases), were attended by slow respiration (20 and 25). The ... temperatures were attended by rapid breathing (3 cases,

† ... one of my pupils, Mr. Thomas D. Parry.

may be associated with a slow pulse, *e.g.* R. 77, P. 72, and, what is more common, slow breathing with a rapid pulse, R. 25, P. 150; R. 20, P. 130 and 110.

The following illustrations of individual cases may exhibit some of the chief of such variations:*

		Quickest pulse.	Respiration.	Slowest pulse.	Respiration.	Mean pulse.	Mean respiration.
I. Case chronic phthisis	M.	[illegible]	72	72	78	87	72†
	E.	124	80	76	96	94	67‡
II. Case acute phthisis	M.	146	60	100	48	120	[illegible]
	E.	148	96	—	—	—	—
III. Case acute phthisis	M.	120	48	[illegible]	40	104	42
	E.	124	60	100	60	110	50
IV. Case acute phthisis	M.	154	76	64	30	106	[illegible]
	E.	154	48	[illegible]	28	106	38
V. Case "low temperatures"	M.	92	32	68	44	78	[illegible]
	E.	—	—	—	—	—	—
VI. Case acute tuberculosis,	M.	136	28	[illegible]	32	112	[illegible]
	E.	152	36	96	32	126	[illegible]
VII. Case "high temperatures."	M.	[illegible]	20	80	[illegible]	97	[illegible]
	E.	104	28	80	20	92	24
VIII. Case chronic phthisis	M.	136	26	90	16	120	25
	E.	—	—	—	—	—	—

In the case of the most rapid pulses the rise of the pulse in the evening is greater in the majority than that of the respiration,§ and this is also to some, but to a less degree, observed in the slowest pulses. It is, however,

* Of these all but No. 5 are females; Nos. 1 and 2 sisters.

† Mean of 27 obs.

‡ Mean of 14 obs.; a girl not hysterical.

§ Quickest pulses (Table 17), respiration from 25 to 40; morning pulse above 100 in 17 out of 33 cases, evening pulse above 100 in 21 out of 26 cases. Respiration from 20 to 25; morning pulse above 100 in 8 out of 12 cases, above 120 in 3 out of 12 cases, evening pulse above 100 in 4 out of 6 cases, and above 120 in 2 out of 6 cases.

scarcely apparent when the means are taken. When in the slowest pulses the pulse remains rapid, the respiration usually does so also,* but the pulse may be slow, both in the morning and evening, while the respiration continues rapid.† The slower pulses are, however, usually associated with some retardation of the respiration, both in the morning and evening, but not usually in the same degree.‡

The pulse-respiration ratio (see Tables 18 and 19) in the majority of cases is found between respiration 1, pulse 3 to 4, both in the quickest pulses and in the means of the pulse to the respiration, but when the pulse is retarded both in the morning and evening the largest proportion is found between resp. 1, pulse 2 to 3. The ratio, when the breathing is rapid, may, however, be absolutely reversed, as pulse 72, resp. 78; pulse 76, resp. 96.§

* Slowest pulses (Table 17).—Morning, pulse above 120 (7 cases), all the respirations are above 35, and all but one above 40, but evening pulse above 120 (19 cases); in five the respirations are above 40, in three 30 to 35, and in one 20 to 25.

† Slowest pulses (Table 17).—Respiration above 40. Morning (19 cases), in 11 only is the pulse above 100 = 57·3 per cent.; evening (14 cases), in 9 = 64·2 per cent. is the pulse above 100.

‡ Thus, slowest pulses (Table 17):

Respiration below 35 morning, 44 cases,	pulse above 90 in only	7 = 15·9	p. c.
„ evening, 35 cases,	„	13 = 37·1	„
Respiration below 30 morning, 33 cases	„	4 = 12·1	„
„ evening, 26 cases	„	7 = 26·9	„
Respiration below 25 morning, 25 cases	„	2 = 8	„
„ evening, 16 cases	„	2 = 125	„

The means of the pulses and respirations give higher proportions of rapid pulses in relation to the lower rates of respiration. Respirations above 35—morning pulse above 100 in 90 per cent., evening pulse above 100 in 81·8 per cent.; morning pulse above 90 in 93·3 per cent., evening pulse above 90 in 95·4 per cent. Respirations below 35—morning pulse above 90 in 43·1 per cent., evening pulse ditto in 62·5 per cent. Respirations below 30—morning pulse above 90 in 59·3 per cent., evening pulse ditto in 43·7 per cent. Respirations below 25—morning pulse above 90 in 42·8 per cent., evening pulse ditto in 22·2 per cent.

§ Dr. Edward Smith, l. c., p. 189, found that the pulse-respiration ratios were on the average not lower than 1 :: 4, nor so high as 1 :: 2·3. The highest varied from 1 :: 1·4 to 1 :: 2·3. The case presenting the lowest 1 from 1 :: 2·8 to 1 :: 5·8. It was raised before a meal, and lowered

A very rapid pulse may also increase the ratio of the pulse to the respirations by more than 6 to 1, but this may also exist to a considerable degree when, without a very rapid pulse, the respiration is disproportionately retarded, and may even be observed in acute tuberculosis.* Such variations of perversion, in either direction, are, however, comparatively speaking, uncommon. (See Table 20.)

The pulse-respiration ratio is, on the whole, very little influenced by the temperature (see Table 21), though a ratio of the pulse to the respiration of less than 2 to 1 is most common in the "low temperatures," inasmuch as the pulse is more influenced by the temperature than the breathing, and a slow pulse and rapid breathing may thus coexist with a low temperature; yet with a high temperature the respiration may rise out of proportion to the pulse.† Ratios of less than 3 to 1 are, among the quickest pulses, most common when the temperature is below 100°, but the same is not observed when the pulse is slow, nor even in the mean pulses, since in the latter a larger percentage both for morning and evening are found above than below a temperature of 100°. When both the pulse and respiration are slow the temperature is usually normal or nearly so, but both a quick pulse and rapid breathing may be simultaneously associated with a low temperature;‡ and,

after a meal, and was highest during sleep at night. Dr. Smith, l.c., p. 192, has, however, observed the very low rates of 1 :: 7 and 1 :: 8 in the earlier stages of phthisis, chiefly in persons of unusual stature.

* Acute tuberculosis, 1 case, slowest pulse, morning 82, respiration 14. Chronic phthisis, 1 case, slowest pulse, morning 90, resp. 16. "Low temperatures," 1 case, quickest pulse, morning 96, resp. 20

† A case of chronic phthisis (see [illegible] Table, p. 440), morning temp. 103·4°, pulse 116, resp. 72. Evening [illegible] 124, resp [illegible] pulse. Also in a case of acute [illegible] 96. Among the slowest pulses [illegible] morning, where, with a temper[illegible] pulse to the respiration [illegible] with a temper[illegible] of acute tu[illegible] phthisis, one [illegible] resp. 36.

‡ Chronic [illegible]

on the other hand, pyrexia may exist with but little acceleration of either, or the temperature may be high and the pulse rapid, while the respiration may be but little accelerated.*

In individual cases also great variations may be noticed on different days in the relations of the pulse and respiration to the temperature, whether the exacerbations of the latter occur in the morning or in the evening, and this is true of all forms of phthisis, being observed in both the acute and chronic types, and also in acute tuberculosis. Evening exacerbations may be attended by a positive diminution in the rapidity of the pulse and breathing, or either of these may be accelerated while the other is retarded, or remains at the same standard as in the morning. In some cases also the pulse falls in the morning more than the temperature, but more in proportion to the temperature than is observed in the respiration; but even when this appears to be the rule, there may be a considerable fall in the evening rate of respiration, while the pulse remains nearly the same and the temperature rises. When exacerbations of temperature occur from evening to morning the pulse and respiration may, nevertheless, fall in frequency, but to a less degree than is most usual where remissions occur; or, under these circumstances, the respiration may be accelerated while the pulse is retarded.

When the temperature falls from morning to evening there is less fall of the pulse and respiration; but here also nearly all possible variations may be observed in their relations of acceleration and retardation. In one case the occurrence of pneumothorax produced marked acceleration of the pulse, but had very little effect on the rate of respiration.

The comparatively definite relation of the pulse to the temperature is in accordance with what is observed in most febrile diseases; but the absence of correspondence between

* Chronic phthisis.—Morning, quickest pulse 120, temp. 104·2°, resp. 28. Evening, pulse 120, temp. 103·3°, resp. 32. Slowest pulse, morning, 88, temp. [illegible] Mean morning pulse 100, temp. 101°, resp. 26. Mean evening [illegible]°, resp. 26.

the degree of pyrexia and the respiration is remarkable.* The rate of respiration is, indeed, chiefly influenced by the mal-aeration of the blood; and this, especially in chronic cases, may be slowly produced by the gradual progress of the disease, the foci of which in an active state may be too limited to excite intense febrile action. Slower rates of respiration may possibly be induced by gradual impoverishment of the blood and wasting of tissues, but I have not observed that they were specially common in cases marked by extreme anæmia or by great emaciation. A rapid pulse with a low temperature is an unfavorable sign, as indicating exhaustion; and the same is probably true of acceleration of the respiration under similar circumstances, as it is sometimes observed out of proportion to the rate of the pulse on the approach of death. Marked perversions of the pulse-respiration ratios in either direction have also an unfavorable significance; but in comparing the two forms in the fatal and non-fatal cases it will be seen that a rapid pulse with comparatively slow breathing is more common in the former than the latter, while high ratios of the respiration to the pulse are most common in the latter (see Table 19, Means). The greater prevalence of the last named under these circumstances is probably due to the patients so affected being for the most part admitted to hospital with recent acute affections, which tend to increase the rapidity of respiration in proportion to the pulse. Rapid pulses with slow breathing have never been found by Dr. E. Smith in the early stages of phthisis.

* Traube, 'Symptomen,' &c., p. 49, classifies as the causes of rapid breathing:

(*a*) Maloxygenation; (*b*) irritation of the fibres of the vagus in the lung; (*c*) elevation of the temperature of the blood; (*d*) pain. He adduces the observations of Sklarek to show that narrowing of the air-passages may retard the rate of respiration.

APPENDIX.

TABLE 1.—*Showing the average frequency with which, during 24 hours, the temperatures indicated were reached, but not exceeded, in all the observations in each class of phthisis.*

Class of cases.	No. of cases.	Average No. of observations in each case.	Subnormal temperatures.	98° to 99°.	99° to 100°.	100° to 101°.	101° to 102°.	102° to 103°.	103° to 104°.	104° to 105°.	105° to 106°.	106° to 107°.
Acute tuberculosis..............................	VIII	15	...	1·3	1·7	7·4	25·4	22·8	27·5	9·9	2·2	...
Acute phthisis	XXV	24	0·9	2·7	11·4	16·9	25·3	23·6	14·5	3·6	...	0·1*
High temperatures. No deaths............	VIII	23·7	1·1	5·1	11·3	25·4	18·4	17·9	16·	3·6	...	...
Chronic phthisis	XVIII	26·7	5·8	7·5	15·9	21·6	22·2	16·6	8·3	0·3	0·2	...
Low temperatures. No deaths	XXI	18·7	...	30·1	43	16·8	7·6	1·4	0·8	...	...	...

* One observation 106·7 in 38 days.

TABLE 2.—*Showing the total number of cases in each class of phthisis, in which—(a) Normal and subnormal temperatures were observed during 24 hours. (b) The cases in which the temperatures indicated were reached,* but not exceeded.*

Class of cases.	No. of cases.	Fell to subnormal.	Fell to (98° to 99°) normal.	Not exceeding 100°.	Not exceeding 101°.	Not exceeding 102°.	Not exceeding 103°.	Not exceeding 104°.	Not exceeding 105°.	Not exceeding 106°.	Not exceeding 107°.
Acute tuberculosis	8	...	1	...	...	...	2	1	3	2	...
		...	12·5	...	...	...	25	12·5	37·5	25·1	...
Acute phthisis............	25	6	8	...	...	3	4	9	8	...	1
		24	32	...	...	12	16	36	32	...	4
High temperatures. No deaths	8	1	2	...	...	...	...	5	3	...	...
		12·5	25	...	...	...	...	62·5	37·5	...	...
Chronic phthisis.........	18	3	6	...	1	2	4	9	1	1	...
		16·6	33·3	...	5·5	11·1	22·2	50	5·5	5·5	...
Low temperatures. No deaths	21	...	20	3	5	9	2	2	...	...	...
		...	90·4	14·2	23·8	42·8	9·5	9·5	...	...	...

* By "reached" is meant that the temperature was higher than the degree lower. *E.g.* "Not exceeding 101°" means that the temperature exceeded 100° but did not exceed 101°. The second lines of figures indicate the percentage of such cases.

TABLE 3.—*Showing the number of cases in which, in 75 cases of different classes of phthisis, normal and subnormal temperatures were attained, and also the percentage of observations in which these were observed in each case.*

	No. of cases.	Less than 5 per cent. of observations.	5 to 10 per cent. of observations.	10 to 20 per cent. of observations.	20 to 30 per cent. of observations.
ACUTE TUBERCULOSIS	VIII	...	...	...	...
E. Pyrexia. M. Normal	2	1	1	...	...
E. Pyrexia. M. Subnormal	...	...	...	...	...
E. Normal. M. Subnormal	...	...	...	...	...
M. Pyrexia. E. Normal	...	...	...	...	...
M. Pyrexia. E. Subnormal	...	...	...	...	...
M. Normal. E. Subnormal	...	...	...	...	...
M. and E. Subnormal	...	...	...	...	...
ACUTE PHTHISIS	XXIV	...	...	...	...
E. Pyrexia. M. Normal	13	6	6	...	1
E. Pyrexia. M. Subnormal	6	5	1	..	...
E. Normal. M. Subnormal	2	1	1	...	...
M. Pyrexia. E. Normal	7	6	...	1	...
M. Pyrexia. E. Subnormal	3	3	...	...	...
M. Normal. E. Subnormal	1	1	...	...	..
M. and E. Subnormal	5	5	...	...	...
HIGH TEMPERATURES	VIII	...	...	...	...
E. Pyrexia. M. Normal	7	4	3	...	...
E. Pyrexia. M. Subnormal	1	1	...	...	...
E. Normal. M. Subnormal	2	2	...	...	...
M. Pyrexia. E. Normal	3	3	...	...	...
M. Pyrexia. E. Subnormal	...	...	...	...	...
M. Normal. E. Subnormal	...	...	...	...	...
M. and E. Subnormal	1	1	...	...	...
CHRONIC PHTHISIS	XV	...	...	...	...
E. Pyrexia. M. Normal	8	5	2	...	1
E. Pyrexia. M. Subnormal	6	6	...	...	...
E. Normal. M. Subnormal	1	...	1	...	...
M. Pyrexia. E. Normal	3	3	...	...	...
M. Pyrexia. E. Subnormal	1	1	...	...	...
M. Normal. E. Subnormal	1	1	...	...	...
M. and E. Subnormal	2	2	...	...	...
LOW TEMPERATURES	XX	...	...	...	...
E. Pyrexia. M. Normal	8	5	1	2	...
E. Pyrexia. M. Subnormal	3	3	...	...	...
E. Normal. M. Subnormal	1	1	...	...	...
M. Pyrexia. E. Normal	6	3	1	2	...
M. Pyrexia. E. Subnormal	1	1	...	...	...
M. Normal. E. Subnormal	2	2	...	...	...
M. and E. Subnormal	...	...	...	...	...

TABLE 4.—*Showing the frequency per cent. of the maximum and average morning and evening temperatures in the whole number of observations in each class of cases of phthisis.*

Class of cases.	Maximum morning temperature.							Maximum evening temperature.									Average morning temperature.					Average evening temperature.					
	98° to 99°.	99° to 100°	100° to 101°.	101° to 102°.	102° to 103°	103° to 104°	104° to 105°	98° to 99°.	99° to 100°.	100° to 101°.	101° to 102°.	102° to 103°.	103° to 104°.	104° to 105°.	105° to 106°.	106° to 107°.	98° to 99°.	99° to 100°.	100° to 101°.	101° to 102°	102° to 103°	98° to 99°.	99° to 100°.	100° to 101°	101° to 102°	102° to 103°.	103° to 104°.
Acute tuberculosis. Deaths. 8 cases..	..	..	..	50	..	25	25	...	...	.	...	12·5	37·5	25	25	...	...	12·5	37·5	37·5	12·5	..	...	12·5	12·5	50	25
Acute phthisis. Deaths. 25 cases	..	.	16	12	20	36	16	..	..	.	16	8	56	16	.	4*	12	16	28	20	24	...	8	32	36	16	8
High temperatures. No deaths. 8 cases	..	..	...	25	50	12·5	12·5	...	.	...	...	...	75	25	.	...	.	62·5	12·5	25	...	..	12·5	50	37·5	...	...
Chronic phthisis. Deaths. 18 cases	5·5	..	5·5	16·6	22·2	50	.	...	...	5·5	..	27·7	55·5	5·5	5·5	...	27·7	16·6	33·3	22·2	...	16·6	11·1	22·2	38·7	11·1	.
Low temperatures. No deaths. 21 cases .	..	23·8	38	23·8	9·5	4·7	..	4·7	28·5	28·5	19	14·2	4·7	...	...	..	52·2	47·1	.		...	28·5	57·1	14·3	.	...	.

* One observation.

TABLE 5.—*Showing the number of cases in each class of phthisis in which the average of the whole number of morning and evening observations in each case did not exceed the temperatures indicated.*

Class of cases.	No. of cases.	Average morning temperatures.					Average evening temperatures.					
		Not exceeding 99°.	Not exceeding 100°.	Not exceeding 101°.	Not exceeding 102°.	Not exceeding 103°.	Not exceeding 99°.	Not exceeding 100°.	Not exceeding 101°.	Not exceeding 102°.	Not exceeding 103°.	Not exceeding 104°.
Acute tuberculosis	8	... ...	1 12·5	3 37·5	3 37·5	1 12·5	... ...	... ...	1 12·5	1 12·5	4 50	2 25
Acute phthisis...	25	3 12	4 16	1 28	5 20	6 24	... ...	2 8	8 32	9 36	4 16	2 8
High temperatures not ending fatally ...	8	... ...	5 62·5	1 12·5	2 25	... ...	... ...	1 12·5	5 62·5	3 37·5	... ...	... ...
Chronic phthisis	18	5 27·7	3 16·6	6 33·3	4 22·2	... ...	2 11·1	3 16·6	4 22·2	7 38·8	2 11·1	... ...
Low temperatures not ending fatally ...	21	11 52·3	10 47·6	... ...	... ...	... ...	6 28·5	12 57·1	3 14·2	... ...	... ...	... ...

TABLE 6.—*Showing the number of cases within pyrexial limits, in which the morning temperature, in each class of phthisis, exceeded that of the preceding or of the subsequent evening, and also the proportion in which this occurred to the whole number of observations in each case.*

Note.—The numbers in this table do not precisely correspond to those of the preceding tables, as in some of the former the excess of the morning over the evening occurred within the normal limits of temperature. One case is also omitted in the class of acute tuberculosis, as the number of observations was too small to allow of a safe estimate of the percentage.

Class of cases.	Total numbers with morning excess.	1 to 10 per cent. of observations.	10 to 20 per cent of observations	20 to 30 per cent. of observations.	30 to 40 per cent of observations.	40 to 50 per cent of observations.	50 to 60 per cent. of observations.	60 to 70 per cent of observations.	70 to 80 per cent of observations.	80 to 90 per cent. of observations
ACUTE TUBERCULOSIS. 7 cases.*										
M. higher than previous E... ..	V	2	...	1	2	...	...	...	...	...
M. higher than subsequent E...	V	1	2	...	1	1	...	...	..	..
ACUTE PHTHISIS. 24 cases.										
M. higher than previous E......	XXI	2	5	6	4	3	...	...	1	..
M. higher than subsequent E...	XXI	3	4	5	3	5	1	...	..	...
HIGH TEMPERATURES. 7 cases.										
M. higher than previous E......	VII	3	2	..	...	1	.	...	1	..
M. higher than subsequent E..	VII	2	3	...	...	...	1	...	1	...
CHRONIC PHTHISIS. 15 cases.										
M. higher than previous E......	XV	2	2	2	5	2	1	...	...	1
M. higher than subsequent E...	XV	2	2	4	4	1	1	1	...	...
LOW TEMPERATURES. 20 cases.										
M. higher than previous E......	XVIII	4	5	4	3	...	1	1	...	..
M. higher than subsequent E...	XVII	4	3	5	2	1	1	...	1	...

Note.—In further explanation of this table it may be stated that remissions from morning to evening and exacerbations from morning to evening were each respectively sometimes limited to a single observation in individual cases. This was observed of the remissions in question in 2 cases of acute tuberculosis, in 5 cases of acute phthisis, in 2 cases of chronic phthisis, in 3 cases of "high temperatures," and in 4 cases of "low temperatures." Exacerbations from evening to morning were similarly confined to a single observation in 2 cases of acute tuberculosis, in 2 cases of acute phthisis, in 2 cases of chronic phthisis, in 3 cases of "high temperatures," and in 4 cases of "low temperatures," and these cases corresponded in all but 4 instances.

* One case omitted, as the number of observations was too small to afford fair percentages in proportion to the remainder.

Table 7.—*Showing the number of cases in each class of phthisis in which the maxima and minima, and also the averages for each case of all the remissions and exacerbations respectively indicated, were observed.*

Note.—The averages are formed by adding together, first, all the exacerbations and all the remissions [redacted] each [redacted] and dividing the result by the number of observations in each.

Class of cases.	Number of cases.	Averages of remissions and exacerbations. Number of cases of each average.						Maxima of remissions and exacerbations. Number of cases of each.										Minima of remissions and exacerbations. Number of cases of each.									
		4° to 5°.	3° to 4°.	2° to 3°.	1° to 2°.	1° to 0·5°.	0·5° to 0°.	8°.	7° to 8°.	6° to 7°.	5° to 6°.	4° to 5°.	3° to 4°.	2° to 3°.	1° to 2°.	1° to 0·5°.	0·5° to 0°.	3°.	2° to 3°.	1° to 2°.	0·8° to 1°.	0·6° to 0·7°.	0·5°.	0·4°.	0·3°.	0·2°.	0·1°.
Acute Tuberculosis ...	VIII																										
Remissions E. to M. ..	8	...	1	1	6	...	..	...	..	.	1	2	3	2	...	...	...	...	1	...	2	...	...	2	...	8	...
Remissions M. to E	5[1]	..	...	..	2	3	..	.	...	...	...	...	.	1	3	1	...	...	...	1	...	1	...	...		2	...
Exacerbations M. to E.	8	...	1	1	5	1	...	...	...	...	...	2	4	1	1	...	...	...	...	1	...	1	...	1	...	4	1
Exacerbations E. to M.	6[2]	...	..	..	2	2	2	..	...		...	...	1	...	3	1	1	...	...	...	1	1	...	...	1	2	1
Acute Phthisis	XXIV																										
Remissions E. to M. ..	24	.	1	5	13	8	2	1	...	1	1	5	6	4	4	2	...	.	...	4	...	3	..	4	...	9	4
Remissions M. to E	22[3]	...	...	2	6	7	7	...	...	...	...	...	3	4	4	6	5	...	1	...	...	...	...	7	..	12	2
Exacerbations M. to E.	24	..	1	2	7	8	2	1	...	2	2	8	4	6	5	...	1	...	...	2	1	2	1	6	...	11	2
Exacerbations E. to M.	21[4]	...	...	2	8	6	5	...	...	...	.	4	2	5	8	1	1	...	...	...	...	5	...	3	...	9	4

HIGH TEMPERATURES (no deaths)....	VIII																										
Remissions E. to M. ..	8	...	...	2	6	1	...	...	...	...	1	2	3	2	...	..	...	...	...	...	1	1	..	1	...	5	...
Remissions M. to E...	8	..	1	...	3	3	1	...	..	...	...	...	2	2	1	1	2	1	..	...	...	...	..	4	...	2	1
Exacerbations M. to E.	8	...	...	3	1	1	...	.	...	...	...	4	2	2	...	...	...	...	...	...	. .	...	.	2	...	4	2
Exacerbations E. to M.	8	.	...	1	2	3	2	...	...	...	...	...	2	1	2	1	2	..	1	...	1	...	...	1	...	3	2
CHRONIC PHTHISIS	XV																										
Remissions E. to M....	15	1	...	2	11	1	...	...	...	1	1	4	2	7	...	...	...	..	1	1	1	1	...	1	...	9	1
Remissions M. to E.	15	...	...	...	5	7	3	...	...	...	...	1	2	2	6	2	2	...	...	...	...	1	1	1	1	8	3
Exacerbations M. to E.	15	...	..	1	11	8	...	...	..	1	...	1	2	8	8	...	...	...	...	1	...	2	..	1	...	10	1
Exacerbations E. to M.	15	...	...	...	9	3	3	..	...	..	...	...	1	3	9	1	1	...	..	..	...	1	1	2	...	8	3
LOW TEMPERATURES (no deaths)	XX																										
Remissions E. to M.	19[5]	...	...	...	7	7	5	...	...	...	1	...	4	5	6	2	1	...	...	...	...	2	...	2	...	10	5
Remissions M. to E...	19[6]	...	...	1	4	8	6	...	...	...	...	...	1	7	5	1	5	...	...	...	. .	4	...	3	1	9	2
Exacerbations M. to E.	19[7]	...	...	1	4	9	5	...	...	...	...	1	8	3	9	2	1	...	...	...	...	1	...	2	1	11	4
Exacerbations E. to M.	18[8]	...	..	...	3	10	5	...	...	...	...	...	2	5	3	4	4	...	...	1	1	3	...	1	...	10	2

[1] 3 cases, no remissions M. to E.=37·5 per cent.
[2] 2 cases, no exacerbations E. to M=25 per cent.
[3] 2 cases, or 8·3 per cent., no remissions M. to E.
[4] 3 cases, or 12·5 per cent., no exacerbations E. to M.

[5] 1 case, or 5 per cent., no remissions E. to M.
[6] 1 case, or 5 per cent., no remissions M. to E.
[7] 1 case, or 5 per cent., no exacerbations M. to E.
[8] 2 cases, or 10 per cent., no exacerbations E. to M.

TABLE 8.—*Showing the percentage of cases in which each of the minima of the remissions and exacerbations indi-*

Class of cases.	No. of cases.	Percentage of cases in which each of the AVERAGES of remissions and exacerbations indicated were observed.						Percentage of cases in sions and exacerbations			
		4° to 5°	3° to 4°	2° to 3°	1° to 2°	0·5° to 1°	0° to 0·5°	6° to 7°	5° to 6°	4° to 5°	3° to 4°
ACUTE TUBERCULOSIS ...	VIII										
Remissions E. to M. ...	8	...	12·5	12·5	75	...	...	...	12·5	25	37·5
Remissions M. to E. ...	5[1]	...	...	...	40	60	16·6	...	...	...	.
Exacerbations M. to E.	8	.	12·5	12·5	62·5	12·5	...	...	..	25	50
Exacerbations E. to M.	6[2]	...	...	...	33·3	33·3	33·3	...	...	...	16·6
ACUTE PHTHISIS	XXIV										
Remissions E. to M. ...	24	...	4·1	20·8	54·1	12·5	8·3	8·3[3]	4·1	20·8	25
Remissions M. to E. ...	22[4]		.	9	27·2	31·8	31·8	...	.	13·6	18·1
Exacerbations M. to E.	24		4·1	8·3	66·6	12·5	8·3	12·5[5]	8·3	12·5	16·6
Exacerbations E. to M.	21[6]	...	..	9·5	38	28·5	23·8	...	...	19	9·5
HIGH TEMPERATURES (no deaths)	VIII										
Remissions E. to M.	8	..	...	25	62·5	12·5	...	.	12·5	25	37·5
Remissions M. to E. ...	8	..	12·5	...	37·5	37·5	12·5	...	..	...	25
Exacerbations M. to E.	8	...	..	37·5	50	0·5	...	..	...	50	25
Exacerbations E. to M.	8	...	...	12·5	25	37·5	25	...	...	...	25
CHRONIC PHTHISIS	XV										
Remissions E. to M. ...	15	6·6	...	13·3	66·6	13·3	...	6·6	6·6	26·5	13·3
Remissions M. to E. ...	15	...	...	...	33	46·2	20	...	...	6·6	13·3
Exacerbations M. to E.	15	...	6·6	72·6	20	...	...	6·6	...	6·6	13·3
Exacerbations E. to M.	15	...	...	...	39·6	39·6	19·8	..	...	...	6·6
LOW TEMPERATURES (no deaths)	XX										
Remissions E. to M. ..	19[8]	...	...	...	36·8	36·8	26·3	...	5·2	...	20·8
Remissions M. to E. .	19[9]	...	...	5·2	20·8	42·1	31·2	...	.	...	5·2
Exacerbations M. to E.	19[10]	..	...	5·2	21·2	47·8	26·5	...	...	5·2	15·6
Exacerbations E. to M.	18[11]	..	.	.	16·8	55·5	27·5	...	.	...	11

[1] 3 cases = 37·5 per cent., no remissions M to E.
[2] 2 cases = 25 per cent., no exacerbations E. to M.
[3] 1 case, a single remission of 8·8°.
[4] 2 cases = 8·3 per cent., no remissions M. to E.
[5] 1 case, a single exacerbation of 8·4°.
[6] 3 cases, 12·5 per cent., no exacerbations E. to M.

averages of the remissions and exacerbations and the maxima and cated were observed. (Companion to Table 7.)

which the MAXIMA of remis- indicated were observed.				Percentage of cases in which the MINIMA of remissions and exacerbations were observed.									
2° to 3°.	1° to 2°.	0·5° to 1°.	0° to 0·5°.	3°.	2° to 3°.	1° to 2°.	0·8 to 1°.	0·6° to 0·7°.	0·5°.	0·4°.	0·3°.	0·2°.	0·1°.
25	...	...	...	...	12·5	25	...	...	...	25	...	37·5	...
20	60	20	...	...	...	20	...	40	...	...	...	40	...
12·5	12·5	...	...	...	...	12·5	...	12·5	...	12·5	...	50	12·5
...	50	16·6	16·6	...	...	...	16·6	16·6	...	...	16·6	33·3	16·6
16·6	16·6	8·2	...	...	...	16·6	...	12·5	...	16·6	...	37·5	16·6
18·1	27·2	22·7	...	...	4·5	...	...	...	...	31	...	54·5	9
25	20·8	...	4·1	...	...	8·3	4·1	8·3	4·1	20·8	...	45·8	8·3
23·8	38·	4·5	4·7	...	...	...	...	23·8	...	14·2	...	42·7	19
25	...	...	...	...	...	...	12·5	12·5	...	12·5	...	62·5	...
25	12·5	12·5	25	12·5[7]	...	...	...	...	...	50	...	25	12·5
25	...	...	...	...	...	...	...	...	...	25	...	50	25
12·5	25	12·5	25	...	12·5	...	12·5	...	...	12·5	...	37·5	25
46·2	...	...	...	...	6·6	6·6	6·6	6·6	...	6·6	...	59·4	6·6
13·3	39·6	13·3	13·3	...	...	...	...	6·6	6·6	6·6	6·6	52·8	20
53·3	20	..	...	...	...	6·6	...	13·3	...	6·6	...	66·6	6·6
20	59·4	6·6	6·6	...	...	...	...	6·6	6·6	13·3	...	52·8	20·
26	31·2	10·4	5·2	...	...	...	...	10·5	...	10·5	...	52·6	26
36·4	26·3	5·2	26·3	...	...	...	...	20·8	...	15·6	5·2	46·8	10·4
15·6	47·8	10·4	5·2	...	...	...	...	5·2	...	10·4	5·2	57·2	20·8
27·5	16·5	22	22	...	...	5·5	5·5	16·5	...	5·5	...	55	11

[7] In 1 case the minimum remission from M. to E. was 3·70.

[8] 1 case = 5 per cent., no remissions E. to M.

[9] 1 case = 5 per cent., no remissions M. to E.

[10] 1 case = 5 per cent., no exacerbations M. to E.

[11] 2 cases = 10 per cent., no exacerbations E. to M.

Table 9.—*Showing the average frequency per cent. of every single of cases in each*

Class of cases.	No. of cases.		Remissions.								
			No. of cases.	6° to 8°.	5° to 6°	4° to 5°	3° to 4°.	2° to 3°	1° to 2°.	0·5° to 1°.	0° to 0·5°.
Acute Tuberculosis	VIII	Remissions E. to M.	8	...	0·9	1·8	13·9	27·4	25·5	14·6	11·2
		Remissions M. to E.	5[1]	...	...	...	...	2	46	37·3	14·6
Acute Phthisis..............	XXIV	Remissions E. to M.	24	1·9	0·7	5·1	5·5	17·4	30·5	20·6	20·5
		Remissions M. to E.	22[2]	...	...	0·9	2·2	6·9	22·6	20·9	45·6
High Temperatures (no deaths)	VIII	Remissions E. to M.	8		1·2	2·8	7·2	16·2	36·9	16·7	18·6
		Remissions M. to E.	8	...	...	...	13·5	12	16·5	2·5	55·3
Chronic Phthisis	XV	Remissions E. to M.	15	2·6	0·6	4·8	4·4	21·8	28·8	20·2	16
		Remissions M. to E.	15	...	...	0·5	2	6·8	24·4	25·1	40·6
Low Temperatures (no deaths)	XX	Remissions E. to M.	19[6]	...	0·5	...	2·2	7·7	26·1	24·6	38·8
		Remissions M. to E.	19[7]	...	...	...	...	7	19·5	30·2	43·6

This table is formed by estimating for each case the percentage of the observations of the frequency with which every single exacerbation and remission occurred. These percentages are then added together for each class, and divided by the number of cases in each class.

remission and exacerbation of temperature occurring in the whole number class of phthisis.

Exacerbations.									
	No. of cases.	6° to 8°.	5° to 6°.	4° to 5°.	3° to 4°.	2° to 3°.	1° to 2°.	0·5° to 1°.	0° to 0·5°.
Exacerbations M. to E.	8	...	...	4·5	16·4	13·7	31·5	11·8	19·3
Exacerbations E. to M.	6[2]	...	...	...	1·7	1·7	9·1	34·1	53·3
Exacerbations M. to E.	24	0·8	0·7	2·2	5·5	14	33·3	21	21·7
Exacerbations E. to M.	21[4]	...	...	1·7	1·6	7·4	36·6	29·9	23·1
Exacerbations M. to E.	8	...	...	6·4	9	18·9	26·6	17·6	20·8
Exacerbations E. to M.	8	...	...	...	2	15·4	21·7	22·7	38
Exacerbations M. to E.	15	0·8	...	0·9	3	14·3	40·2	21·4	17·7
Exacerbations E. to M.	15	...	0·2	0·2	1·8	4·2	37·3	18·5	37·4
Exacerbations M. to E.	19[6]	...	...	0·6	1·4	6	29·2	20·7	42·7
Exacerbations E. to M.	18[8]	...	...	...	3	2·7	21·5	32·2	42

[1] In 3 cases no remissions M. to E.
[2] In 2 cases no exacerbations E. to M.
[3] In 2 cases no remissions M. to E.
[4] In 3 cases no exacerbations E. to M.
[5] In 1 case no remissions E. to M.
[6] In 1 case no exacerbations M. to E.
[7] In 1 case no remissions M. to E.
[8] In 2 cases no exacerbations E. to M.

TABLE 10.—*Showing the relative number of cases in which the maxima, and also the averages of the remissions and exacerbations, in any given case, differed or corresponded, comparing the remissions from evening to morning with the exacerbations from morning to evening, and the remissions from morning to evening with the exacerbations from evening to morning.*

Classes of phthisis	Remissions from evening to morning compared with exacerbations from morning to evening.							Remissions from morning to evening compared with exacerbations from evening to morning						
	Number of cases.	Maximum of remissions from evening to morning compared with maximum of exacerbations from morning to evening.			Averages of remissions from evening to morning compared with averages of exacerbations from morning to evening			Number of cases	Maximum of remissions from morning to evening compared with maximum of exacerbations from evening to morning			Averages of remissions from morning to evening compared with averages of exacerbations from evening to morning		
		Max. rem. E. to M. greater than max. exac. M. to E.	Max. rem. E. to M. equal to max. exac. M. to E.	Max. rem. E. to M. less than max. exac. M. to E.	*Av. rem. E. to M. greater than av. exac. M. to E.*	*Av. rem. E. to M. equal to av. exac. M. to E.*	*Av. rem. E. to M. less than av. exac. M. to E.*		Max. rem. M. to E. greater than max. exac. E. to M.	Max. rem. M. to E. equal to max. exac. E. to M.	Max. rem. M. to E. less than max. exac. E. to M.	*Av. rem. M. to E. greater than av. exac. E. to M.*	*Av. rem. M. to E. equal to av. exac. E. to M.*	*Av. rem. M. to E. less than av. exac. E. to M.*
Acute tuberculosis...	VIII	3	5	...	1	7	...	V	1	2	2	1	3	1
Acute phthisis	XXIV	5	13	6	3	18	3	XX	4	9	7	3	9	8
High temperatures..	VIII	2	3	3	2	5	1	VIII	3	3	2	3	3	2
Chronic phthisis ...	XV	7	7	1	5	9	1	XV	4	5	6	3	5	7
Low temperatures...	XVIII	8	4	6	3	12	3	XVIII	4	11	3	7	6	5
Totals	LXXIII	25	32	16	14	51	8	LXVI	16	30	20	17	26	23

From the table it would appear that in the larger number of cases there is no close correspondence between the extent of the remissions as compared with the exacerbations which might be supposed to follow them. The explanation appears to lie in the irregularity of the interspersion of morning exacerbations and evening remissions; but it appears almost impossible to estimate the total effect of these except on analyses of the general temperatures.

Thus—

The maximum remissions from evening to morning were greater than the maximum exacerbations from morning to evening	in 34·2	per cent.	of the cases.
Equal	in 43·8	„	„
Less than	in 21·9	„	„
The averages of the whole number of remissions in individual cases from evening to morning were greater than the similar averages of the exacerbations from morning to evening	in 19·1	„	„
Equal	in 69·8	„	„
Less than	in 10·9	„	„
The maximum remissions from morning to evening were greater than the maximum exacerbations from evening to morning	in 24·2	„	„
Equal	in 45·4	„	„
Less than	in 30·3	„	„
The averages of the remissions from morning to evening were greater than the averages of the exacerbations from evening to morning	in 25·7	„	„
Equal	in 49·0	„	„
Less than	in 34·8	„	„

TABLE 11.—*Showing the quickest and slowest pulses, and also the*

Class of cases.	No. of cases.	Quickest pulses observed.											
		Pulse 170 to 180.	Pulse 160 to 170	Pulse 150 to 160.	Pulse 140 to 150.	Pulse 130 to 140.	Pulse 120 to 130.	Pulse 110 to 120.	Pulse 100 to 110.	Pulse 90 to 100	Pulse 80 to 90.	Pulse 70 to 80.	Pulse 60 to 70.
ACUTE TUBERCULOSIS—													
Morning	7	...	..	2	2	1	2	...	...	..	...	...	...
Evening	6	1	...	2	...	...	3	...	...	...	...	...	...
ACUTE PHTHISIS—													
Morning	23	...	1	2	8	2	5	1	4	...	...	..	...
Evening	19	...	...	2	8	1	3	2	3	...	..	...	...
CHRONIC PHTHISIS—													
Morning	21	..	...	2	4	1	8	2	3	1	...	...	...
Evening	16	..	...	...	2	3	6	1	4	...	..	...	...
HIGH TEMPERATURES—													
Morning	7	...	...	...	1	1	...	1	4	...	...	...	...
Evening	4	..	...	...	1	...	1	...	1	...	1	...	...
LOW TEMPERATURES—													
Morning	16	...	...	...	..	1	1	2	4	2	6	...	...
Evening	9	...	...	...	1	...	1	...	2	1	2	1	1
TOTALS—													
Morning	LXXIV	...	1	6	15	6	16	6	15	3	6	...	...
Evening	LIV	1	...	4	12	4	14	3	10	1	3	1	1

means of all the pulses, observed in seventy-four cases of phthisis.

Slowest pulses observed.										No. of cases.	Mean of pulses observed.									
Pulse 140 to 150.	*Pulse 130 to 140*	*Pulse 120 to 130.*	*Pulse 110 to 120.*	*Pulse 100 to 110.*	*Pulse 90 to 100.*	*Pulse 80 to 90.*	*Pulse 70 to 80.*	*Pulse 60 to 70.*	*Pulse 50 to 60.*		Pulse 140 to 150.	Pulse 130 to 140	Pulse 120 to 130	Pulse 110 to 120	Pulse 100 to 110	Pulse 90 to 100	Pulse 80 to 90	Pulse 70 to 80	Pulse 60 to 70.	Pulse 50 to 60
	..	2	...	...	1	4	..	..	.	7	.	2	1	2	1	1		..	..	..
	.	2	1	..	1	1	1	..		5		1	1	2	1	.	.	..		..
	—	3	..	5	4	8	1	2	..	23	.	1	7	8	8	9	1	.	..	
1	...	2	3	3	3	6	.	1	...	15	.	1	2	6	4	..	2	.	..	...
.	2		1	4	2	7	3	2	..	21	1	1	8	8	[illegible]	· 4	4	...	...	.
..	2	2	3	1	1	3	2	2	...	14	1	1	8	1	2	4	1	1	...	..
..	..	..	...	.	1	3	1	1	1	7	..	...	...	...	1	6	.	...	..	...
..	...	...	.	...	1	3	...	..	...	3	..	..	...	1	1	1	...	.	...	..
..	...	...	..	...	2	3	2	6	3	16	...	...	...	...	8	2	8	5	3	...
.	...		...		1	1	2	4	1	9	.	.	.	1	1	1	8	..	[illegible]	1
...	2	[illegible]	1	9	10	25	7	11	4	LXXIV	1	4	11	8	18	16	8	5	8	...
1	2	6	7	4	7	14	5	7	1	XLVI	1	8	6	11	9	[illegible]	6	1	2	1

TABLE 12.—*Showing the frequency per cent. of pulses of the frequency the means of the pulses, in*

Class of cases.	Quickest pulses.				Slowest			
	No. of cases.	Above 140.	Above 120.	Above 100.	Above 140.	Above 120.	Above 100.	Below 100.
Acute Tuberculosis—								
Morning	7	57·1	100	100	...	28·5	28·5	71·4
Evening	6	50	100	100	...	33·3	50	50
Acute Phthisis—								
Morning	23	47·8	78·2	100	...	13	34·7	65·2
Evening	19	52·7	73·6	100	5·2	15·7	47·3	52·7
Chronic Phthisis—								
Morning	21	28·5	71·4	95·2	...	9·5	33·3	66·7
Evening	16	12·5	68·7	100	...	25	50	50
High Temperatures (not fatal)—								
Morning	7	14·2	25·5	100	...	...	...	100
Evening	4	25	50	75	...	...	...	100
Low Temperatures (not fatal)—								
Morning	16	6·2	12·5	50	...	...	...	100
Evening	9	11·1	22·2	44	...	...	...	100
Totals—								
Morning	LXXIV	29·7	59·4	87·8	...	9·4	22·9	77·1
Evening	LIV	31·4	64·8	90·7	1·8	16·6	37	63

indicated, occurring in the quickest and slowest observed, and also of seventy-four cases of phthisis.

pulses.				Means of pulses.								
Below 90.	Below 80.	Below 70.	Below 60.	No. of cases.	Above 140.	Above 120.	Above 100.	Below 100.	Below 90.	Below 80.	Below 70.	Below 60.
57·1	...	..	...	7	...	42·8	85·7	14·2	14·2	...	..	...
33·3	16·6	...	...	5	...	40	100	...	...	...	..	...
47·8	13	8·6	...	23	..	34·7	82·6	17·4	4·3	...	..	...
36·8	5·2	5·2	...	15	...	20	86·6	13·3	13·3	..	...	...
57·1	23·7	9·5	...	21	4·7	9·5	61·9	[redacted]	19	...	...	...
43·7	25	12·5	...	14	7·1	35·7	57·1	42·8	14·2	7·1	...	...
85·7	42·8	14·2	14·2	7	...	..	14·2	85·7	..	...	...	...
75	...	...	...	3	..	...	66·6	33·3	...	...	...	...
87·5	68·7	56·2	18·7	16	...	...	18·7	81·2	68·7	50	18·7	...
88·8	77·7	66·6	33·3	9	...	...	22·2	77·7	66·6	33·3	33·3	11·1
63·5	29·7	20·2	5·4	LXXIV	1·3	21·6	56·7	43·2	21·6	10·8	4·	...
50	24	14·8	1·8	XLVI	2·1	45·6	65·2	34·8	21·7	8·6	6·5	2·1

TABLE 13.—*Showing the number of cases in which given pulses coincided with the temperatures indicated, for the quickest, the slowest, and the means of the pulses observed in seventy-four cases of phthisis.*

Note.—In the column of "means" the temperatures recorded are the means of those observed throughout the case for the morning and evening respectively.

Quickest pulses, morning.

Temperatures.	No. of cases.	Pulse 170 to 180	Pulse 160 to 170	Pulse 150 to 160	Pulse 140 to 150	Pulse 130 to 140	Pulse 120 to 130	Pulse 110 to 120	Pulse 100 to 110	Pulse 90 to 100	Pulse 80 to 90.	Pulse 70 to 80	Pulse 60 to 70.
105° to 106°	...												
104° to 105°	2						1		1				
103° to 104°	6			2	1		1	2					
102° to 103°	8		1		3		1	1	2				
101° to 102°	19			3	3	2	5		5	1			
100° to 101°	11			1	2	2	1	1	3	1			
99° to 100°	16				4	1	5	2	2	1	1		
98° to 99°	11				1	1	2		2		5		
97° to 98°	1				1								
96° to 97°	..												
95° to 96°	...												
Totals...	74	...	1	6	15	6	16	6	15	3	6	...	...

Slowest pulses, morning

Temperatures.	*No. of cases.*	*Pulse 140 to 150.*	*Pulse 130 to 140.*	*Pulse 120 to 130.*	*Pulse 110 to 120.*	*Pulse 100 to 110.*	*Pulse 90 to 100*	*Pulse 80 to 90.*	*Pulse 70 to 80.*	*Pulse 60 to 70.*	*Pulse 50 to 60.*
105° to 106°	..										
104° to 105°	..										
103° to 104°	.										
102° to 103°	5				1	2		2			
101° to 102°	8			3		2	1	1	1		
100° to 101°	13			1		1	3	5	1	2	
99° to 100°	24		2	1		3	2	10	3	2	1
98° to 99°	18					1	3	6	1	5	2
97° to 98°	4						1		1	1	1
96° to 97°	1							1			
95° to 96°	1									1	
Totals...	74	..	2	5	1	9	10	25	7	11	4

Mean of pulses and mean temperatures, morning

Temperatures.	No. of cases.	Pulse 140 to 150	Pulse 130 to 140	Pulse 120 to 130	Pulse 110 to 120	Pulse 100 to 110	Pulse 90 to 100	Pulse 80 to 90.	Pulse 70 to 80	Pulse 60 to 70.	Pulse 50 to 60.
105° to 106°	...										
104° to 105°	...										
103° to 104°	...										
102° to 103°	6			3	2	1					
101° to 102°	16	1	3	3	2	4	3				
100° to 101°	15			4	1	5	3	2			
99° to 100°	23				2	5	10	2	4		
98° to 99°	14		1	1	1	3		4	1	3	
97° to 98°	.										
96° to 97°	...										
95° to 96°	.										
Totals...	74	1	4	11	8	18	16	8	5	3	...

Quickest pulses, evening

Temperatures.	No. of cases.	Pulse 170 to 180.	Pulse 160 to 170.	Pulse 150 to 160.	Pulse 140 to 160.	Pulse 130 to 140.	Pulse 120 to 130.	Pulse 110 to 120.	Pulse 100 to 110.	Pulse 90 to 100.	Pulse 80 to 90	Pulse 70 to 80.	Pulse 60 to 70.
105° to 106°	...	...	...	...	...	...	...	...	...	...	...	...	...
104° to 105°	3	1	...	...	1	...	1	...	...	...	...	...	...
103° to 104°	9	...	...	2	2	1	4	...	...	...	...	...	...
102° to 103°	12	...	...	1	5	1	3	1	1	...	...	...	...
101° to 102°	14	...	...	1	...	...	2	2	9	...	...	...	...
100° to 101°	10	...	...	...	3	...	3	...	...	...	3	1	...
99° to 100°	3	...	...	...	1	1	...	...	...	...	...	...	1
98° to 99°	2	...	...	...	...	...	1	...	...	1	...	...	...
97° to 98°	...	...	...	...	...	...	...	...	...	...	...	...	...
96° to 97°	...	...	...	...	...	...	...	...	...	...	...	...	...
95° to 96°	1	...	...	...	...	1	...	...	...	...	...	...	...
Totals ...	54	1	...	4	12	4	14	3	10	1	3	1	1

Slowest pulses, evening.

Temperatures.	*No. of cases.*	*Pulse 140 to 150*	*Pulse 130 to 140.*	*Pulse 120 to 130.*	*Pulse 110 to 120.*	*Pulse 100 to 110.*	*Pulse 90 to 100.*	*Pulse 80 to 90.*	*Pulse 70 to 80.*	*Pulse 60 to 70*	*Pulse 50 to 60.*
105° *to* 106°	1	...	...	...	...	...	...	...	1	...	...
104° *to* 105°	1	...	...	...	1	...	...	...	...	...	...
103° *to* 104°	5	1	1	1	1	...	...	1	...	...	...
102° *to* 103°	5	...	...	1	2	...	1	1	...	...	...
101° *to* 102°	11	...	...	4	...	1	2	2	1	1	...
100° *to* 101°	9	...	1	...	2	1	1	3	...	1	...
99° *to* 100°	10				...	1	3	3	1	2	...
98° *to* 99°	9				1			3	1	3	1
97° *to* 88°	1	...	...	...	...	...	...	1	...	...	...
96° *to* 97°	2	...	...	...	...	1	...	...	1	...	...
95° *to* 96°	...	...	...	...	...	...	...	...	...	...	...
Totals ...	54	1	2	6	7	4	7	14	5	7	1

Mean of pulses and mean temperatures, evening.

Temperatures.	No. of cases.	Pulse 140 to 160.	Pulse 130 to 140.	Pulse 120 to 130.	Pulse 110 to 120.	Pulse 100 to 110.	Pulse 90 to 100.	Pulse 80 to 90.	Pulse 70 to 80.	Pulse 60 to 70.	Pulse 50 to 60.
105° to 106°	...	...	...	...	...	...	...	...	...	...	...
104° to 105°	...	...	...	...	...	...	...	...	...	...	...
103° to 104°	2	...	...	...	2	...	...			...	...
102° to 103°	8	...	2	2	1	2	1	...		...	...
101° to 102°	12	...	1	2	3	4	2		...	...	...
100° to 101°	12	1	...	1	3	2	1	4		...	...
99° to 100°	8	...	...	1	1		2	1	1	2	
98° to 99°	4		...	...	1	1	...	1		...	1
97° to 98°	...	...	...	...	...	...	...	...	...	...	...
96° to 97°	...	...	...	...	...	...	...	...	...	...	...
95° to 96°	...	...	...	...	...	...	...		...	...	
Totals...	46	1	3	6	11	9	6	6	1	2	1

TABLE 14.—*Showing the frequency of the respirations corresponding to the quickest and slowest pulses, and also the mean frequency of the respiration, in seventy-four cases of phthisis.*

Respirations corresponding to quickest pulses.

Class of cases.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
ACUTE TUBERCULOSIS—											
Morning	7		.	1	2	2		1	1	.	
Evening	6			3		1	2		.		
ACUTE PHTHISIS—											
Morning	23	1	2	5	4	2	4	2	1	2	
Evening	19	1	1	..	7	3	1	1	6	.	.
CHRONIC PHTHISIS—											
Morning	21	1	...	1	..	5	3	3	6	1	1
Evening	16	1		.		5	6	2	2	2	..
HIGH TEMPERATURES—											
Morning	7		...		...		1	2	1	3	.
Evening	4			.	.			3	1	.	.
LOW TEMPERATURES—											
Morning	16			..	1	1	2	4	2	6	
Evening	9			...		2		1	2	4	
TOTALS—											
Morning	74	2	2	7	7	10	10	12	11	12	1
Evening	54	2	1	3	7	9	9	7	10	6	

Respirations corresponding to slowest pulses

Class of cases	*No. of cases*	*Respiration above 70.*	*Respiration 60 to 70.*	*Respiration 50 to 60.*	*Respiration 45 to 50.*	*Respiration 40 to 45.*	*Respiration 35 to 40.*	*Respiration 30 to 35.*	*Respiration 25 to 30.*	*Respiration 20 to 25.*	*Respiration below 20*
ACUTE TUBERCULOSIS—											
Morning	7	..	1	2		1	..	1	..	1	1
Evening	6	.	.	1	.	2	..	1	.	1	1
ACUTE PHTHISIS—											
Morning	23	.			3	5	5	4	2	3	1
Evening	19	1	1	.	1	3	2	3	5	2	1
CHRONIC PHTHISIS—											
Morning	21	1	.	..		5	2	4	1	6	2
Evening	16	1	.			4	2	2	3	4	..
HIGH TEMPERATURES—											
Morning	7	.	...	..		.	1		3	3	
Evening	4							2		2	
LOW TEMPERATURES—											
Morning	16					1	3	2	2	7	1
Evening	9		..	.	.		1	1	2	5	..
TOTALS—											
Morning	74	1	1	2	3	12	11	11	8	20	5
Evening	54	2	1	1	1	9	5	9	10	14	2

Mean of all respirations.

Class of cases.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
ACUTE TUBERCULOSIS—											
Morning	7		...	2	..	1	1	1	1	1	.
Evening	5		...	1		...	1	2	1		...
ACUTE PHTHISIS—											
Morning	23		...	1	3	3	9	3	1	2	1
Evening	15	.	..	1	2	1	7	1	1	1	1
CHRONIC PHTHISIS—											
Morning	21	1	...	.		2	4	4	8	1	1
Evening	14	.	1	.	.	1	5	3	3	1	.
HIGH TEMPERATURES—											
Morning	7		.			...	1		4	2	..
Evening	3	..	..	..		..	..	1		2	...
LOW TEMPERATURES—											
Morning	16	..		..	.	1	1	4	4	6	1
Evening	9	..	..		.	..	2	1	2	4	...
TOTALS—											
Morning	74	1	.	3	3	7	18	12	18	11	3
Evening	46	..	1	2	2	2	16	8	7	8	1

TABLE 15.—*Showing the percentages of cases in which the mean frequency of respiration was observed in different classes of phthisis.*

Class of cases.	No. of cases.	Respiration above 40.	Respiration above 30.	Respiration below 25.
ACUTE TUBERCULOSIS—				
Morning	7	42·8	71·4	1·4
Evening	5	20	80	...
ACUTE PHTHISIS—				
Morning	23	30·4	82·6	13·
Evening	15	26·6	80	13·3
CHRONIC PHTHISIS—				
Morning	21	14·2	52·3	9·5
Evening	14	14·2	71·4	7·1
HIGH TEMPERATURES—				
Morning	7	...	14·2	28·5
Evening	3	...	33·3	66·6
LOW TEMPERATURES—				
Morning	16	6·2	37·5	37·5
Evening	9	...	33·3	44·4
TOTALS—				
Morning	LXXIV	18·9	56·7	18·9
Evening	XLVI	15·2	65·2	19·5

Table 16.—*Showing the number of cases in which respirations of the frequency indicated corresponded with the temperatures indicated, and also the correspondence between the means of the respirations and the means of the temperatures, in seventy-four cases of phthisis.*

Respirations corresponding to morning temperatures accompanying quickest pulses.											
Temperatures.	No. of cases.	Respiration above 70.	Respiration 60 to 70	Respiration 50 to 60	Respiration 45 to 50	Respiration 40 to 45.	Respiration 35 to 40	Respiration 30 to 35	Respiration 25 to 30	Respiration 20 to 25.	Respiration below 20.
105° to 106°	..										
104° to 105°	2								1	1	
103° to 104°	6	1		1		2	1			1	
102° to 103°	8		1	3		1	1	1	1		
101° to 102°	19	1	1	1		3	2	4	5	2	
100° to 101°	11			1		1	2	3		3	1
99° to 100°	16				6	2	2	1	3	2	
98° to 99°	11				1	1	2	3	1	3	
97° to 98°	1			1							
96° to 97°											
95° to 96°	..										
Totals...	74	2	2	7	7	10	10	12	11	12	1

Respirations corresponding to morning temperatures accompanying slowest pulses											
Temperatures.	*No. of cases.*	*Respiration above 70.*	*Respiration 60 to 70.*	*Respiration 50 to 60*	*Respiration 45 to 50*	*Respiration 40 to 45*	*Respiration 35 to 40.*	*Respiration 30 to 35.*	*Respiration 25 to 30*	*Respiration 20 to 25*	*Respiration below 20.*
105° to 106°											
104° to 105°											
103° to 104°											
102° to 103°	5					1	1	2		1	
101° to 102°	8			1	1	3	1			1	1
100° to 101°	13		1		1	1	3	1	1	5	
99° to 100°	24	1		1	1	3	2	5	5	4	2
98° to 99°	18					4	2	2		8	2
97° to 98°	4						1		2	1	
96° to 97°	1						1				
95° to 96°	1							1			
Totals ..	74	1	1	2	3	12	11	11	8	20	5

Mean number of respirations compared with mean temperature—morning.											
Temperatures.	No of cases.	Respiration above 70	Respiration 60 to 70	Respiration 50 to 60	Respiration 45 to 50	Respiration 40 to 45	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
105° to 106°	...										
104° to 105°	..										
103° to 104°											
102° to 103°	6				3		2		1		
101° to 102°	16			3		2	5	1	4	1	
100° to 101°	15					3	4	2	3	2	1
99° to 100°	23					1	3	5	8	5	1
98° to 99°	14	1				1	2	4	2	3	1
97° to 98°	..										
96° to 97°	...										
95° to 96°	...										
Totals...	74	1	.	3	3	7	16	12	18	11	3

Respirations corresponding to evening temperatures accompanying quickest pulses.										
Temperatures.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
105° to 106°											
104° to 105°	3			1			1	1			
103° to 104°	9	1		1	1	1	1	2	1	1	
102° to 103°	12	1			4	3	1	1	2		
101° to 102°	14			1	1	2	3	1	5	1	
100° to 101°	10		1			3	2	1	1	2	
99° to 100°	3				1			1		1	
98° to 99°	2								1	1	
97° to 98°											
96° to 97°											
95° to 96°	1						1				
Totals	54	2	1	3	7	9	9	7	10	6	

Respirations corresponding to evening temperatures accompanying slowest pulses.										
Temperatures	*No. of cases*	*Respiration above 70*	*Respiration 60 to 70.*	*Respiration 50 to 60.*	*Respiration 45 to 50.*	*Respiration 40 to 45*	*Respiration 35 to 40.*	*Respiration 30 to 35.*	*Respiration 25 to 30.*	*Respiration 20 to 25*	*Respiration below 20.*
105° to 106°	1										1
104° to 105°	1					1					
103° to 104°	5	1				1	1		1	1	
102° to 103°	5					1		2	2		
101° to 102°	11			1	1	2		3	2	2	
100° to 101°	9					1	2	3	1	1	1
99° to 100°	10	1	1			1			1	6	
98° to 99°	9					2	1		2	4	
97° to 98°	1								1		
96° to 97°	2						1	1			
95° to 96°											
Totals	54	2	1	1	1	9	5	9	10	14	2

Mean number of respirations compared with mean temperature—evening.										
Temperatures	No. of cases.	Respiration above 70	Respiration 60 to 70.	Respiration 50 to 60.	Respirat on 45 to 50.	Respiration 40 to 45.	Resp ration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
105° to 106°											
104° to 105°											
103° to 104°	2						1	1			
102° to 103°	8			1	1		2	2	2		
101° to 102°	12				1	2	4	3	1	1	
100° to 101°	12			1			5		2	3	1
99° to 100°	8	1					2	2		3	
98° to 99°	4						1		2	1	
97° to 98°											
96° to 97°											
95° to 96°											
Totals	46	1		2	2	2	15	8	7	8	1

TABLE 17.—*Showing the number of cases in which respirations and pulses of the frequency indicated concurred, and also in which means of respirations corresponded with means of pulses in seventy-four cases of phthisis.*

Respirations corresponding to quickest pulses—morning.

Pulse.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
170 to 180											
160 to 170	1			1							
150 to 160	6	1				4			1		
140 to 150	15		2	4	3	1	4	1			
130 to 140	6			1			1	1	2	1	
120 to 130	16			1	3	3	1	3	3	2	
110 to 120	6	1				1	1		1	2	
100 to 110	16				1	1	2	4	3	3	1
90 to 100	3							2		1	
80 to 90	6						1	1	1	3	
70 to 80											
60 to 70											
50 to 60											
Totals ...	74	2	2	7	7	10	10	12	11	12	1

Respirations corresponding to slowest pulses—morning.

Pulse.	*No. of cases*	*Respiration above 70.*	*Respiration 60 to 70.*	*Respiration 50 to 60.*	*Respiration 45 to 50.*	*Respiration 40 to 45.*	*Respiration 35 to 40.*	*Respiration 30 to 35.*	*Respiration 25 to 30.*	*Respiration 20 to 25.*	*Respiration below 20.*
170 *to* 180											
160 *to* 170											
150 *to* 160											
140 *to* 150											
130 *to* 140	2					2					
120 *to* 130	5		1	1	1	1	1				
110 *to* 120	1							1			
100 *to* 110	9				1	4	1	2	1		
90 *to* 100	10				1	2	4		1	1	1
80 *to* 90	25			1		1	2	6	3	10	2
70 *to* 80	7	1				1	1		1	3	
60 *to* 70	11					1	1	2	1	5	1
50 *to* 60	4						1		1	1	1
Totals...	74	1	1	2	3	12	11	11	8	20	5

Mean of respirations compared with means of pulses—morning.

Pulse.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
170 to 180											
160 to 170											
150 to 160											
140 to 150	1					1					
130 to 140	4			3		1					
120 to 130	11				3	2	4	1	1		
110 to 120	8						5	1	2		
100 to 110	18					2	6	8	2		
90 to 100	16						1	1	8	5	1
80 to 90	8	1							2	3	2
70 to 80	5					1			2	2	
60 to 70	3							1	1	1	
50 to 60											
Totals ...	74	1	...	3	3	7	16	12	18	11	3

Respirations corresponding to quickest pulses—evening.

Pulse.	No. of cases.	Respiration above 70	Respiration 60 to 70	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
170 to 180	1			1							
160 to 170											
150 to 160	4			1	2		1				
140 to 150	12	1			4	3	2	2			
130 to 140	4				1	1	2				
120 to 130	14	1	1	1		2	1	4	2	2	
110 to 120	8					1	1		1		
100 to 110	10					1	2		6	1	
90 to 100	1								1		
80 to 90	3					1		1		1	
70 to 80	1									1	
60 to 70	1									1	
50 to 60											
Totals ...	54	2	1	3	7	9	9	7	10	6	...

Respirations corresponding to slowest pulses—evening

Pulse.	*No. of cases.*	*Respiration above 70.*	*Respiration 60 to 70*	*Respiration 50 to 60*	*Respiration 45 to 50.*	*Respiration 40 to 45*	*Respiration 35 to 40.*	*Respiration 30 to 35.*	*Respiration 25 to 30*	*Respiration 20 to 25*	*Respiration below 20.*
170 *to* 180											
160 *to* 170											
150 *to* 160											
140 *to* 150	1	1									
130 *to* 140	2					1		1			
120 *to* 130	6			1	1	1		2		1	
110 *to* 120	7					2	2		3		
100 *to* 110	4		1			1	1		1		
90 *to* 100	7					2		3	1	1	
80 *to* 90	14					1	1	2	3	7	
70 *to* 80	5	1				1		1		1	1
60 *to* 70	7						1		2	3	1
50 *to* 60	1									1	
Totals ..	54	2	1	1	1	9	5	9	10	14	2

Mean of respirations compared with means of pulses—evening.

Pulse.	No. of cases.	Respiration above 70	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
170 to 180											
160 to 170											
150 to 160											
140 to 150	1						1				
130 to 140	3			1			2				
120 to 130	6				1		1	4			
110 to 120	11			1	1	1	5	3			
100 to 110	9					1	3	1	3	1	
90 to 100	6		1				2		2	1	
80 to 90	6								2	3	1
70 to 80	1									1	
60 to 70	2						1			1	
50 to 60	1									1	
Totals ...	46	...	1	2	2	2	15	8	7	8	1

Table 18.—*Showing the frequency with which the pulse-respiration ratios of the means of all the*

Quickest pulses.									Slowest	
Class of cases.	No. of cases.	Respiration 1. Pulse 0–1	Respiration 1 Pulse 1–2	Respiration 1 Pulse 2–3	Respiration 1 Pulse 3–4	Respiration 1 Pulse 4–5	Respiration 1 Pulse 5–6	Respiration 1. Pulse 6–7	*Class of cases.*	*No. of cases.*
Acute Tuberculosis—									Acute Tuberculosis—	
Morning	7	...	...	2	4	...	1	...	*Morning* . . .	7
Evening	6	..	...	3	2	1	...	...	*Evening*	6
Acute Phthisis—									Acute Phthisis—	
Morning	23	...	..	9	7	4	3	...	*Morning*	23
Evening	19	...	1	2	10	6	...	...	*Evening*	19
Chronic Phthisis—									Chronic Phthisis—	
Morning . ..	21	.	1	2	9	5	2	2	*Morning*	21
Evening	16	.	1	1	10	8	1	...	*Evening*	16
High Temperatures—									High Temperatures—	
Morning	7	..	...	...	3	2	1	1	*Morning*	7
Evening	4	...	...	1	2	1	...	...	*Evening*	4
Low Temperatures—									Low Temperatures—	
Morning	16	...	...	3	8	4	1	...	*Morning*	16
Evening	9	...	..	3	4	1	1	...	*Evening*	9
Totals—									Totals—	
Morning ..	74		1	16	31	15	8	3	*Morning*	74
Evening .	54	..	2	10	28	12	2	—	*Evening*	54

ratios indicated occurred in seventy-four cases of phthisis, and also the pulses and respirations observed.

pulses.							Means of pulse to means of respiration.								
Respiration 1. Pulse 0—1	*Respiration 1. Pulse 1—2.*	*Respiration 1. Pulse 2—3*	*Respiration 1. Pulse 3—4*	*Respiration 1. Pulse 4—5.*	*Respiration 1. Pulse 5—6.*	*Respiration 1. Pulse 6—7.*	Class of cases.	No. of cases.	Respiration 1. Pulse 0–1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
							ACUTE TUBERCULOSIS—								
...	2	3	1	...	...	1	Morning	7	...	...	3	2	2	...	...
...	...	3	2	1	...	..	Evening	5	..	...	1	3	1	...	...
							ACUTE PHTHISIS—								
...	...	15	7	1	..	...	Morning .. .	23	..	...	6	14	2	1	..
...	2	7	5	5	...	...	Evening	15	.	...	2	10	3	..	...
							CHRONIC PHTHISIS—								
1	2	5	8	4	1	...	Morning	21	...	1	1	14	4	1	...
1	1	4	4	5	1	...	Evening	14	...	1	3	7	3	..	...
							HIGH TEMPERATURES—								
...	...	5	1	1	...	...	Morning	7	...	...	1	4	2	...	...
...	...	1	1	2	..	...	Evening	3	...	...	...	2	1	...	...
							LOW TEMPERATURES—								
...	2	6	7	1	...	...	Morning	16	...	1	6	7	2	...	...
...	1	3	4	1	...	...	Evening	[illegible]	...	1	3	4	1	...	...
							TOTALS—								
1	6	34	24	7	1	1	Morning	74	..	2	17	41	12	2	...
1	4	18	16	14	1	.	Evening	46	.	2	9	26	9	.	...

Table 19.—*Showing the percentage frequency of the pulse-respiration and*

Quickest pulses.							*Slowest*	
Class of cases.	No. of cases.	Respiration 1 Pulse less than 2.	Respiration 1 Pulse 2—3	Respiration 1 Pulse 3—4.	Respiration 1 Pulse 4—5	Respiration 1 Pulse more than 5.	*Class of cases.*	*No. of cases.*
Acute Tuberculosis—							Acute Tuberculosis—	
Morning	7	...	28·5	55·5	...	14·2	*Morning* .. .	7
Evening	6	...	50	33·3	16·6	...	*Evening*	6
Acute Phthisis—							Acute Phthisis—	
Morning	23	.	39	30·3	17·3	13	*Morning* . . .	23
Evening	19	5·2	10·3	52·6	31·5	..	*Evening*	19
Chronic Phthisis—							Chronic Phthisis—	
Morning	21	4·7	9·5	42·8	23·8	19	*Morning*	21
Evening	16	6·2	6·2	62·5	18·7	6·2	*Evening*	16
High Temperatures—							High Temperatures—	
Morning	7	.	...	42·8	28·5	28·5	*Morning*	7
Evening	4	...	25	50	25	...	*Evening*	4
Low Temperatures—							Low Temperatures—	
Morning	16	...	18·7	50	25	6·2	*Morning*	16
Evening .. .	9	.	33·3	44·4	11·1	11·1	*Evening* .	9
Totals—							Totals—	
Morning ...	74	1·3	21·6	41·8	20·2	14·8	*Morning*	74
Evening . .	54	3·7	18·5	51·8	22·2	3·7	*Evening*	54

ratios indicated and also of the ratios of the means of the pulses respirations.

pulses.					Means of pulse to means of respiration.						
Respiration 1. Pulse less than 2.	*Respiration 1. Pulse 2—3.*	*Respiration 1. Pulse 3—4.*	*Respiration 1. Pulse 4—5.*	*Respiration 1. Pulse more than 5.*	Class of cases.	No. of cases.	Respiration 1. Pulse less than 2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse more than 5.
					ACUTE TUBERCULOSIS—						
28·5	42·8	14·2	...	14·2	Morning	7	—	42·8	28·5	28·5	..
...	50	33·3	16·6	...	Evening	5	...	20	60	20	...
					ACUTE PHTHISIS—						
...	65·1	30·3	4·3	..	Morning	23	...	26	60·7	8·6	4·3
10·5	36·7	26·3	26·3	..	Evening	15	...	13·3	66·6	19·9	..
					CHRONIC PHTHISIS—						
14·2	23·8	38·0	19·0	4·7	Morning	21	4·7	4·7	66·6	19	4·7
12·5	25	25	31·2	6·2	Evening	14	7·1	21·4	50	21·4	...
					HIGH TEMPERATURES—						
...	71·4	14·2	14·2	...	Morning	7	...	14·2	55·5	28·5	...
...	25	25	50	...	Evening	3	...	...	66·6	33·3	...
					LOW TEMPERATURES—						
12·5	37·5	43·7	6·2	...	Morning	16	6·2	37·5	43·7	12·5	...
11·1	33·3	44·4	11·1	...	Evening	9	11·1	33·3	44·4	11·1	...
					TOTALS						
9·4	45·9	32·4	9·4	2·7	Morning	74	2·7	22·9	55·3	15·9	2·7
9·2	33·3	29·6	25·9	1·8	Evening	46	4·3	19·5	56·4	19·5	...

TABLE 20.—*Showing the correspondence between pulses of the frequency indicated and the pulse-respiration ratios indicated.*

Quickest pulse—morning.								
Pulse.	No. of cases.	Respiration 1, Pulse 0—1	Respiration 1, Pulse 1—2	Respiration 1, Pulse 2—3	Respiration 1, Pulse 3—4	Respiration 1, Pulse 4—5	Respiration 1, Pulse 5—6	Respiration 1, Pulse 6—7
170 to 180	...	.	.	.	...	..	.	...
160 to 170	1	.	..	..	1	..	..	..
150 to 160	6	.	..	1	4	...	1	...
140 to 150	15	..	..	4	5	5	1	...
130 to 140	6	.	..	1	1	1	2	1
120 to 130	16	...	...	5	5	4	1	1
110 to 120	6	...	1	...	2	2	1	...
100 to 110	15		...	3	7	3	1	1
90 to 100	3	.	.	..	2	...	1	..
80 to 90	6	...	..	2	4	...	...	...
70 to 80	...	...	...	...	...	...	...	...
60 to 70	...	...	...	...	...	...	...	...
50 to 60	...	...	...	...	...	..	..	...
Totals...	74	.	1	16	31	15	8	3

Slowest pulse—morning.								
Pulse.	*No. of cases*	*Respiration 1, Pulse 0—1.*	*Respiration 1, Pulse 1—2.*	*Respiration 1, Pulse 2—3*	*Respiration 1, Pulse 3—4*	*Respiration 1, Pulse 4—5*	*Respiration 1, Pulse 5—6*	*Respiration 1, Pulse 6—7*
170 *to* 180	.	.	...	...	...	..	.	.
160 *to* 170		..	...	.	.	.	.	..
150 *to* 160	..	..	..	..	.	..	..	
140 *to* 150		..	..	..	.	.	.	
130 *to* 140	2	..	...	..	2	..	.	.
120 *to* 130	5	..	1	2	2	..	.	.
110 *to* 120	1	.	.	..	1	.	.	...
100 *to* 110	9	..	...	6	2	1	.	..
90 *to* 100	10	..	..	7	2	..	1	..
80 *to* 90	25		1	11	6	6	.	1
70 *to* 80	7	1	1	2	3	..	...	..
60 *to* 70	11	..	2	4	5	..	..	.
50 *to* 60	4		1	2	1	.	..	..
Totals...	74	1	6	34	24	7	1	1

Means of pulse compared with ratio of means of pulses and respirations—morning.								
Pulse.	No. of cases.	Respiration 1, Pulse 0—1.	Respiration 1, Pulse 1—2	Respiration 1, Pulse 2—3	Respiration 1, Pulse 3—4	Respiration 1, Pulse 4—5	Respiration 1, Pulse 5—6	Respiration 1, Pulse 6—7
170 to 180				..	.	.		.
160 to 170		.				...	..	.
150 to 160	.	.	.		.			
140 to 150	1	.		.	1	..		...
130 to 140	4	.	.	3	1	..		...
120 to 130	11	..	..	3	7	1		.
110 to 120	8		..		6	2	.	...
100 to 110	18	..	..	5	11	2	..	.
90 to 100	16	.	.	2	8	4	2	.
80 to 90	8	..	1	.	4	3	..	
70 to 80	5	.	1	1	3	...	.	...
60 to 70	3	.	.	3	.	.	.	...
50 to 60	..	..	..	...	.	.	.	..
Totals...	74		2	17	41	12	2	..

Quickest pulse—evening.

Pulse.	No. of cases.	Respiration 1. Pulse 0—1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
170 to 180	1	..	...	...	1	...	..	.
160 to 170	..	.	..	...	...	...	...	.
150 to 160	4	..	..	1	2	1	...	...
140 to 150	12	..	1	..	8	8	..	
130 to 140	4	...	..	1	3	...	...	..
120 to 130	14	...	1	3	4	4	2	.
110 to 120	8	.	...	..	2	1	...	.
100 to 110	10	.	...	2	6	3	..	.
90 to 100	1	...	.		1	...	..	..
80 to 90	3	.	..	2	1	..	...	.
70 to 80	1	.	...	...	1	..	..	.
60 to 70	1	..	...	1	...	...	..	.
50 to 60	..	...	..	..	...	...	...	..
Totals...	54	..	2	10	28	12	2	..

Slowest pulse—evening.

Pulse.	*No. of cases.*	*Respiration 1. Pulse 0—1.*	*Respiration 1. Pulse 1—2.*	*Respiration 1. Pulse 2—3.*	*Respiration 1. Pulse 3—4.*	*Respiration 1. Pulse 4—5.*	*Respiration 1. Pulse 5—6.*	*Respiration 1. Pulse 6—7.*
170 *to* 180	.	...	...	...	..		...	.
160 *to* 170	..	...	..	..	..	...	...	.
150 *to* 160			...	...	.	.	...	.
140 *to* 150	1	.	1	..	..	.	.	.
130 *to* 140	2	..	...	..	1	1	...	
120 *to* 130	6	.	..	3	.	2	[illegible]	
110 *to* 120	7		..	2	2	3	...	..
100 *to* 110	4	..	1	1	1	1	.	.
90 *to* 100	7	.	...	2	4	1	...	..
80 *to* 90	14		...	5	5	4	..	..
70 *to* 80	5	1	1	1	1	1	.	
60 *to* 70	7	..	1	3	2	1	..	..
50 *to* 60	1	..	.	1	..	..	..	.
Totals ..	54	1	4	18	16	14	1	.

Means of pulse compared with means of pulse-respiration ratios—evening.

Pulse.	No. of cases.	Respiration 1. Pulse 0—1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
170 to 180	...	..	..	...	..	...	...	...
160 to 170	.		..	..	.	.	.	.
150 to 160		..		.	.	.		...
140 to 150	1	.	.	...	1	..	.	..
130 to 140	3			1	2		.	.
120 to 130	6	...	.	1	2	3	..	...
110 to 120	11	.	..	1	9	1	...	...
100 to 110	9	.		2	4	3	..	...
90 to 100	6	..	1	2	3	.	.	..
80 to 90	6	.	..	...	4	2	..	..
70 to 80	1	..		.	1	.	..	...
60 to 70	2	..	1	1			..	..
50 to 60	1		..	1	...	..	...	..
Totals..	46	..	2	9	26	9	...	...

TABLE 21.—*Showing the correspondence between the temperatures and pulse-respiration ratios indicated in seventy-four cases of phthisis.*

Temperatures and pulse-respiration ratios accompanying quickest pulses—morning.								
Temperatures.	No. of cases.	Respiration 1. Pulse 0—1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
105° to 106°	...	...	...	...	...	...	...	...
104° to 105°	2	...	...	...	...	2	...	...
103° to 104°	6	...	1	...	3	1	1	...
102° to 103°	8	...	...	2	5	1	...	...
101° to 102°	19	...	...	4	7	4	3	1
100° to 101°	11	...	...	1	4	2	3	1
99° to 100°	16	...	...	5	6	3	1	1
98° to 99°	11	...	...	3	6	2	...	...
97° to 98°	1	...	...	1	...	...	...	...
96° to 97°	...	...	...	...	...	...	...	...
95° to 96°	...	...	...	...	...	...	...	...
Totals ...	74	...	1	16	31	15	8	3

Temperatures and pulse-respiration ratios accompanying slowest pulses—morning.								
Temperatures.	*No. of cases.*	*Respiration 1. Pulse 0—1.*	*Respiration 1. Pulse 1—2.*	*Respiration 1. Pulse 2—3.*	*Respiration 1. Pulse 3—4.*	*Respiration 1. Pulse 4—5.*	*Respiration 1. Pulse 5—6.*	*Respiration 1. Pulse 6—7.*
105° *to* 106°	...	...	...	...	...	...	...	...
104° *to* 105°	...	...	...	...	...	...	...	...
103° *to* 104°	...	...	...	...	...	...	...	...
102° *to* 103°	5	...	...	3	2	...	...	...
101° *to* 102°	8	...	...	5	2	...	...	1
100° *to* 101°	13	...	3	6	2	2	...	...
99° *to* 100°	24	1	2	8	9	3	1	...
98° *to* 99°	18	...	...	9	7	2	...	...
97° *to* 98°	3	...	...	1	2	...	...	...
96° *to* 97°	2	...	1	1	...	...	...	...
95° *to* 96°	1	...	...	1	...	...	...	...
Totals ...	74	1	6	34	24	7	1	1

Means of temperatures compared with the means of the pulse-respiration ratios—morning.								
Temperatures.	No. of cases.	Respiration 1. Pulse 0—1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
105° to 106°	...	...	...	...	...	...	...	...
104° to 105°	...	...	...	...	...	...	...	...
103° to 104°	...	...	...	...	...	...	...	...
102° to 103°	6	...	...	3	2	1	...	...
101° to 102°	16	...	...	4	9	3	...	...
100° to 101°	15	...	...	4	6	4	1	...
99° to 100°	23	...	1	3	15	3	1	...
98° to 99°	14	...	1	3	9	1	...	...
97° to 98°	...	...	...	...	...	...	...	...
96° to 97°	...	...	...	...	...	...	...	...
95° to 96°	...	...	...	...	...	...	...	...
Totals ...	74	...	2	17	41	12	2	...

Temperatures and pulse-respiration ratios accompanying quickest pulses—evening.								
Temperatures	No. of cases.	Respiration 1. Pulse 0—1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
105° to 106°	..	...	...	...	..	..	..	..
104° to 105°	3	.	..	..	2	1	..	..
103° to 104°	9	.	1	1	4	2	1	..
102° to 103°	12	...	1	1	8	2	...	..
101° to 102°	14	.	...	3	7	4	...	..
100° to 101°	10	...	...	3	5	2	...	.
99° to 100°	3	...	...	2	...	1	..	.
98° to 99°	3	...	..	..	1	...	1	...
97° to 98°	.	.	...	...	.	..	.	.
96° to 97°	..	..	...	..	..	.	...	..
95° to 96°	..	..	...	...	1	.	..	...
Totals	54		2	10	28	12	2	.

Temperatures and pulse-respiration ratios accompanying slowest pulses—evening.								
Temperatures.	*No. of cases.*	*Respiration 1. Pulse 0—1.*	*Respiration 1. Pulse 1—2.*	*Respiration 1. Pulse 2—3.*	*Respiration 1. Pulse 3—4.*	*Respiration 1. Pulse 4—5.*	*Respiration 1. Pulse 5—6.*	*Respiration 1. Pulse 6—7.*
105° to 106°	1	...	...	...	...	1	...	...
104° to 105°	1	...	...	1	...	...	...	...
103° to 104°	5	...	1	1	1	1	1	...
102° to 103°	5	...	...	2	...	3	...	...
101° to 102°	11	...	1	5	3	2	..	...
100° to 101°	9	...	...	2	4	3	...	...
99° to 100°	10	1	1	2	3	3	...	..
98° to 99°	9	..	1	4	3	1	..	...
97° to 98°	1	..	...	...	1	...	...	...
96° to 97°	2	...	...	1	1	...	.	...
95° to 96°	..	...	...	...	...	...	...	.
Totals ..	54	1	4	18	16	14	1	

Means of temperatures compared with the means of the pulse-respiration ratios—evening.								
Temperatures.	No. of cases.	Respiration 1. Pulse 0—1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
105° to 106°	...	.	..	..	...	...	...	..
104° to 105°	..	.		..	...	...	..	...
103° to 104°	2	..	..	...	2	...	...	..
102° to 103°	8	.	...	1	5	2	..	. .
101° to 102°	12	..	..	4	6	2	...	...
100° to 101°	12	..	.	1	8	3	...	...
99° to 100°	8	...	2	2	2	2	...	...
98° to 99°	4	...	...	1	3	...	...	...
97° to 98°	...	...	...	..	..	...	...	...
96° to 97°	...	...	.	...	. .	..	...	...
95° to 96°	...	.	..	...	...	...	...	...
Totals	46	...	2	9	26	9	..	..

Table 16.—*Showing the number of cases in which respirations of the frequency indicated corresponded with the temperatures indicated, and also the correspondence between the means of the respirations and the means of the temperatures, in seventy-four cases of phthisis.*

Respirations corresponding to morning temperatures accompanying quickest pulses.											
Temperatures.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
105° to 106°		...	...	...	...			...		...	...
104° to 105°	2	...	...	...	...	...		...	1	1	...
103° to 104°	6	1	...	1	...	2	1	...	...	1	...
102° to 103°	8	...	1	3	...	1	1	1	1	...	...
101° to 102°	19	1	1	1		3	2	4	5	2	...
100° to 101°	11	...	...	1	...	1	2	3	...	3	1
99° to 100°	16	...	...	...	6	2	2	1	3	2	...
98° to 99°	11	...	...	...	1	1	2	3	1	3	...
97° to 98°	1	...	...	1	...	...	...	...	...	...	...
96° to 97°	...	...	...	...	...	...	...	...	...	...	...
95° to 96°	...	...	...	...	...	...	...	...	...	...	...
Totals...	74	2	2	7	7	10	10	12	11	12	1

Respirations corresponding to morning temperatures accompanying slowest pulses											
Temperatures.	*No. of cases*	*Respiration above 70.*	*Respiration 60 to 70*	*Respiration 50 to 60*	*Respiration 45 to 50.*	*Respiration 40 to 45.*	*Respiration 35 to 40*	*Respiration 30 to 35.*	*Respiration 25 to 30*	*Respiration 20 to 25*	*Respiration below 20.*
105° to 106°	...	...		...	...		...	...	...	...	...
104° to 105°		...		...	...		...	...	...	...	...
103° to 104°		...								...	
102° to 103°	5		...		...	1	1	2	...	1	...
101° to 102°	8	...	...	1	1	3	1	...	...	1	1
100° to 101°	13	...	1	...	1	1	3	1	1	5	...
99° to 100°	24	1	...	1	1	3	2	5	5	4	2
98° to 99°	18	...	...	...	...	4	2	2	...	8	2
97° to 98°	4	...	...	...	...	...	1	...	2	1	...
96° to 97°	1	...	...	...	...	...	1	...	...	...	...
95° to 96°	1	...	...	...	...	...	...	1	...	...	...
Totals...	74	1	1	2	3	12	11	11	8	20	5

Mean number of respirations compared with mean temperature—morning.											
Temperatures.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
105° to 106°	...	...	...	...		...	...	...		...	...
104° to 105°	...	...	...	...	...	...	...	...	...	...	...
103° to 104°		...	...	...	...	...	...	...	...	...	...
102° to 103°	6			...	3	...	2	...	1		...
101° to 102°	16		...	3	...	2	5	1	4	1	
100° to 101°	15			...		3	4	2	3	2	1
99° to 100°	23	...	...			1	3	5	8	5	1
98° to 99°	14	1		...	...	1	2	4	2	3	1
97° to 98°	...		...	...	...	...	...	...	...	...	...
96° to 97°	...	...	...	...	...	...	...	...	...	...	...
95° to 96°	...	...	...	...	...	...	...	...	...	...	...
Totals...	74	1	..	3	3	7	16	12	18	11	3

Respirations corresponding to evening temperatures accompanying quickest pulses.										
Temperatures.	No. of cases.	Respiration above 70.	Respiration 60 to 70.	Respiration 50 to 60.	Respiration 45 to 50.	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
105° to 106°											
104° to 105°	3			1			1	1			
103° to 104°	9	1		1	1	1	1	2	1	1	
102° to 103°	12	1			4	3	1	1	2		
101° to 102°	14			1	1	2	3	1	5	1	
100° to 101°	10		1			3	2	1	1	2	
99° to 100°	3				1			1		1	
98° to 99°	2								1	1	
97° to 98°											
96° to 97°											
95° to 96°	1						1				
Totals	54	2	1	3	7	9	9	7	10	6	–

Respirations corresponding to evening temperatures accompanying slowest pulses.											
Temperatures	*No. of cases*	*Respiration above 70.*	*Respiration 60 to 70*	*Respiration 50 to 60.*	*Respiration 45 to 50.*	*Respiration 40 to 45*	*Respiration 35 to 40.*	*Respiration 30 to 35.*	*Respiration 25 to 30.*	*Respiration 20 to 25.*	*Respiration below 20.*
105° to 106°	1										1
104° to 105°	1					1					
103° to 104°	5	1				1	1		1	1	
102° to 103°	5					1		2	2		
101° to 102°	11			1	1	2		3	2	2	
100° to 101°	9					1	2	3	1	1	1
99° to 100°	10	1	1			1			1	6	
98° to 99°	9					2	1		2	4	
97° to 98°	1								1		
96° to 97°	2						1	1			
95° to 96°											
Totals...	54	2	1	1	1	9	5	9	10	14	2

Mean number of respirations compared with mean temperature—evening.											
Temperatures.	No. of cases.	Respiration above 70.	Respira'on 60 to 70	Respiration 50 to 60.	Respiration 45 to 50	Respiration 40 to 45.	Respiration 35 to 40.	Respiration 30 to 35.	Respiration 25 to 30.	Respiration 20 to 25.	Respiration below 20.
105° to 106°											
104° to 105°											
103° to 104°	2						1	1			
102° to 103°	8			1	1		2	2	2		
101° to 102°	12				1	2	4	3	1	1	
100° to 101°	12			1			5		2	3	1
99° to 100°	8	1					2	2		3	
98° to 99°	4						1		2	1	
97° to 98°											
96° to 97°											
95° to 96°											
Totals ..	[illegible]	1	...	2	2	2	15	8	7	8	1

ratios indicated occurred in seventy-four cases of phthisis, and also the pulses and respirations observed.

pulses.							Means of pulse to means of respiration.								
Respiration 1. Pulse 0—1.	*Respiration 1. Pulse 1—2.*	*Respiration 1. Pulse 2—3.*	*Respiration 1. Pulse 3—4.*	*Respiration 1. Pulse 4—5.*	*Respiration 1. Pulse 5—6.*	*Respiration 1. Pulse 6—7.*	Class of cases.	No. of cases.	Respiration 1. Pulse 0—1.	Respiration 1. Pulse 1—2.	Respiration 1. Pulse 2—3.	Respiration 1. Pulse 3—4.	Respiration 1. Pulse 4—5.	Respiration 1. Pulse 5—6.	Respiration 1. Pulse 6—7.
							ACUTE TUBERCULOSIS—								
...	2	3	1	...	..	1	Morning	7	...	...	3	2	2	...	...
...	...	3	2	1	...	..	Evening	5	...	..	[illegible]	3	1	..	..
							ACUTE PHTHISIS—								
...	...	15	7	1	..	...	Morning .. .	23	..	...	6	14	2	1	..
...	2	7	5	5	...	...	Evening	15	...	..	2	10	3	...	...
							CHRONIC PHTHISIS—								
1	2	5	8	4	1	...	Morning	21	..	1	1	14	4	1	..
1	1	4	4	[illegible]	1	...	Evening	14	..	1	3	7	[illegible]	...	...
							HIGH TEMPERATURES—								
...	...	5	1	1	...	..	Morning	7	...	...	1	4	2	...	...
...	...	1	1	2	...	...	Evening	3	...	...	...	2	1	...	...
							LOW TEMPERATURES—								
...	2	6	7	1	...	...	Morning	16	...	1	6	7	2	...	...
...	1	3	4	1	...	...	Evening	9	...	1	3	4	1	...	...
							TOTALS—								
1	6	34	24	7	1	1	Morning	74	...	2	17	41	12	2	...
1	4	18	16	14	1	...	Evening . .	46	.	2	9	26	9	..	...